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EDITORIAL

CAUSES OF ASPHYXIAL DEATH

ASPHYXIA has been described as, "a deficiency of available oxygen in the circulating blood, terminating fatally," or more simply, "oxygen want," or as described by Henderson, "asphyxiation is the process that occurs whenever it becomes difficult for the tissues to obtain their normal supply of oxygen. Any absolute deficit kills quickly. As exclusion of oxygen extinguishes a fire, so too in man or animal a deficiency of oxygen in the blood and tissues induces death by asphyxia."

The causes of asphyxial death may be approached from one of two viewpoints: from that of experimental physiology, and from that of clinical medicine.

The approach from the point of view of experimental physiology complicated by biochemical and pathologic variations indicates a wide etiologic field tied together by the common bond of "oxygen want." This approach was elaborated by Coryllos who in his recent article on asphyxia quotes from Meakins and Davis, seven causes of oxygen want:

1. Insufficient partial pressure of oxygen in the inspired air (high altitudes).

2. Obstruction of air passages or resistance to respiration.

3. Obstruction to the passage of gases from the alveoli to the blood (edema, inflammation, exudation, destructive lesions, emphysema).

4. Alteration of oxygen-carrying capacity of the blood (anemia, carbon monoxide, nitrate or chlorate poisoning).

5. Circulatory failure.

6. Pollution of the oxygenated blood by its mixing with the venous blood (uneven ventilation of the lungs, unaerated channels, congenital cardiopathies, and the like).

7. Inhibition of oxydative processes in the tissues (cyanide poisoning).

The approach from the viewpoint of clinical medicine proposes an integration of clinical diseases and accidents, tied together by the common bond of "the relief of oxygen want," or a common form of treatment which when correctly applied is calculated to save the life of the patient. When such clinical relief is not available, or is inefficiently applied the patient succumbs from asphyxiation, a typical example of asphyxial death.

The purpose of bringing about such clinical integration is to emphasize the importance of the combined causes of asphyxia and by such emphasis to bring to bear organized measures for prevention

and relief. When the various causes of asphyxiation have been assembled as a single generic problem a major medical condition is found to exist.

The Committee on Asphyxia of the American Medical Association reporting to the Board of Trustees lists the following causes of asphyxiation (*J. A. M. A.*, May 1, 1937).

1. Asphyxia neonatorum.
2. Asphyxia from gases used industrially.
   (*a*) Carbon monoxide from illuminating gas and from engine exhaust.
   (*b*) Refrigerants such as ammonia, carbon dioxide and dry ice.
   (*c*) Fumes in the manufacture of chemicals.
   (*d*) Gases associated with the oil industry.
   (*e*) Gases in the mining industry.
   (*f*) Fumigation for disease; the destruction of rodents on board ship and elsewhere.
3. Asphyxia from gases in warfare.
4. Asphyxia from drugs, hypnotics, narcotics, and sedatives, including acute alcoholism.
5. Asphyxia from disease, such as acute pulmonary conditions, asthma and cardiac decompensation.
6. Asphyxia from developmental and mechanical abnormalities, such as neonatal atelectasis and collapse of the lung.
7. Asphyxia from anesthesia due to overdosage, idiosyncrasy or a failure to meet mechanical obstruction, occurring in relaxation.
8. Asphyxia from submersion (drowning).
9. Asphyxia from flying at high altitudes.
10. Asphyxia from fire fighting (smoke, chemical poisoning)
11. Asphyxia from obstruction by foreign bodies.
    (*a*) Material caught in the esophagus or inhaled.
    (*b*) Tumors or infections within or without the airway.
12. Asphyxia from electrocution.
13. Asphyxia from strangulation.
14. Asphyxia from allergy.
15. Asphyxia from terminal poliomyelitis.

In an attempt to revise this list and to propose a group of causes of asphyxial death, generally acceptable to recognized authorities in pathology, toxicology, physiology, medical jurisprudence and internal medicine, a communication was directed to a group of twelve men, each distinguished in his particular field. Nine replies resulted, and six favored the listing submitted. One suggested "one other cause of asphyxiation, which is rare, but which might be included. There have been a few deaths from a peculiar form of polyneuritis with facial diplegia in which death has occurred from respiratory failure and asphyxiation."

One correspondent objected that in view of the definition of asphyxial death as that type of asphyxiation which when relieved by suitable means of resuscitation results in saving the life of the patient, and the generally accepted first treatment as artificial respiration, the wisdom of including numbers 14, 11 and 5 could be questioned. "No. 5, Asphyxia from Allergy, certainly should not be treated first with artificial respiration, but rather with intravenous or intracardiac injections of epinephrine solution. No. 11, Asphyxia from Obstruction by Foreign Bodies, in some cases should be treated by artificial respiration, in others this would be likely to do harm rather than good. Of the diseases grouped under No. 5 you would probably kill your patient doing the artificial respiration. [No. 5 includes asthma, which also properly comes under No. 14."

It is the intention of our recommendations for the treatment of asphyxia to precede or accompany artificial respiration in each case with a definite attempt to remove the cause of asphyxiation.

In the case of foreign body obstruction, artificial respiration, per se, if practiced by exposure of the airway, removal of foreign matter, and in the extreme case, intubation and insufflation of oxygen, would dispose of the difficulty in the routine course of treatment.

In allergy, and in other conditions involving circulatory complications, it is the intention to accompany the treatment of acute asphyxia in which the airway is cleared and oxygen administered, by synchronous intravenous or intracardiac medication.

Three authorities answered the communication with diverging opinions. One stated:

"I would define asphyxia as a condition in which absorption of oxygen from the lungs is suddenly and dangerously impaired. It may be brought about by lack of oxygen in the respired air, by occlusion of the respiratory passages or alveoli, or by paralysis of the muscles of respiration. I find it difficult to define asphyxia in its relation to treatment. I would not be inclined to include deficient oxygenation associated with drugs, etc. (4); disease (5); anaesthesia, etc. (7); or allergy (14); unless in conformity with the definition above. For example, the diminution of lung substance by tuberculosis, even when extreme, does not, it seems to me, produce a condition properly designated as asphyxia, whereas, obviously, pulmonary hemorrhage may. I should not designate the condition of oxygen deficiency associated with cardiac decompensation as asphyxia. I do not understand exactly what is meant by asphyxia from allergy, unless it is associated with asthmatic attacks."

We have followed the definition of asphyxia as it appears in Stedman's Medical Dictionary. It is understood that the loss of function of the respiratory center enervating the respiratory mechanism is one of the important causes of asphyxia. Under this heading appears the action of various drugs which are respiratory depressants. From the point of view of the clinician, the immediate problem is oxygen want and the immediate treatment is to supply this oxygen deficiency. Where oxygen is administered over a long period the effects of central depression from drugs may be overcome and the patient rescued. We see this situation in suicidal attempts with barbiturates, morphine, and other drugs as well as in idiosyncrasy to medication, preceding anesthesia. There is a record of a case in which 87 gr. of sodium amytal had been taken. Intratracheal insufflation of oxygen over a sufficient period carried the patient through his central depression. In circulatory abnormalities, if a period of oxygen want can be bridged over to recovery of the cardiovascular mechanism, the patient may be expected to survive.

As a result of the inquiry undertaken, the following list of the causes of asphyxia has been found generally acceptable to the advisory group to which it was referred.

1. Asphyxia Neonatorum.
2. Asphyxia from gases, used industrially.
   - (*a*) Carbon monoxide from illuminating gas and from engine exhaust, coke and coal fires.
   - (*b*) Refrigerants such as ammonia, carbon dioxide and dry ice.
   - (*c*) Fumes in the manufacture of chemicals.
   - (*d*) Gases associated with the oil industry.
   - (*e*) Gases in the mining industry.
   - (*f*) Fumigation for disease; the destruction of rodents on board ship and elsewhere.
3. Asphyxia from gases in warfare.
4. Asphyxia from drugs, hypnotics, narcotics, and sedatives including acute alcoholism.
5. Asphyxia from disease, such as acute pulmonary conditions, asthma and cardiac decompensation.
6. Asphyxia from developmental and mechanical abnormalities, such as neonatal atelectasis and collapse of the lung.
7. Asphyxia from anesthesia due to overdosage, idiosyncrasy or a fail-

ure to meet mechanical obstruction, occurring in relaxation.

8. Asphyxia from submersion (drowning).
9. Asphyxia from high altitude flying.
10. Asphyxia from fire fighting (smoke, chemical poisoning).
11. Asphyxia from obstruction by foreign bodies.
    (*a*) Material caught in the esophagus or inhaled.
    (*b*) Tumors or infections within or without the airway.
12. Asphyxia from electrocution.
13. Asphyxia from strangulation, garroting, hanging.
14. Asphyxia from allergy.
15. Asphyxia from terminal poliomyelitis.
16. Asphyxia from suffocation, overlying, suffocation with soft materials such as pillows, etc.
17. Asphyxia from external pressure on chest and abdomen as caught between two moving objects (automobiles, elevators, and laundry machinery).
18. Asphyxia from collapse of buildings, earth mounds, caves, sand, fire coal in large coal bins, land slides.
19. Asphyxia in manholes, wells, and declivities in the ground, usually from lack of oxygen and increase of CO_2 and sometimes poisonous gases.
20. Asphyxia from polyneuritis, with facial diplegia.

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CORRECTION

Dr. W. Wayne Babcock has called our attention to an unfortunate error in his article on "Experiences with Resection of the Colon and the Elimination of Colostomy" which appeared in the October issue of The American Journal of Surgery. On page 188, where instructions are given for spinal anesthesia, the amount of pontocaine is incorrectly stated as 100 mg., rather than 10 *mg.* The larger amount would of course be highly dangerous for intradural injection.

ORIGINAL ARTICLES

WATER BALANCE*

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THE maintenance of the body fluid level, both intracellular and extracellular, is termed water balance. In a normal person this is at a fairly constant level and is self-regulatory. The kidneys have more to do with the maintenance of the chemical concentration of the plasma,[1] and thus of the entire body fluid, than of the fluid level. Water balance is a complex problem. This paper will deal mainly with the two chief components—water metabolism and salt metabolism. Before abnormal states of water and salt metabolism can be discussed, an understanding of their rôle in health is necessary.

WATER METABOLISM

In health water is ingested by mouth and absorbed from the gastrointestinal tract. It is lost by vaporization, urination and in the stool. As the body needs more water the sensation of thirst reminds one to take more fluid. The fluid absorbed from the gastrointestinal tract is distributed in the body to the blood plasma, to the cells and to the intercellular tissue. The sources of body fluid are (1) water ingested, (2) food ingested, and (3) oxidation of body material.

As expected, the greatest source of water is that taken by mouth, which normally varies from 800 to 3,000 c.c. daily, being greatest during warm weather. Under unusual circumstances the water intake may be greatly increased. Men working in the canyon, building the Boulder Dam, where the temperature was as high as 120° Fahrenheit, drank as much as 10,000 to 18,000 c.c. of water daily. Men working about steel furnaces and in boiler rooms likewise drink huge quantities of water. In such instances the excessive salt loss must be replaced, a problem the industrial medical directors are solving.

The second and very important source of water is from food, since about 70 per cent of its weight is water. The average hospital maintenance diet yields 1,000 to 1,500 c.c. of water, while the soft diet yields about 500 c.c. of water.[2]

The third source of body water is from the oxidation of the body tissue. The oxidation of 1 Gm. of protein yields 0.4 Gm. of water, of 1 Gm. of carbohydrate 0.6 Gm. of water and of fat 1.07 Gm. of water. This source is responsible for from 200 to 400 c.c. of water daily and in starvation as much as 500 c.c. of water becomes available. The combined water content of solid food ingested and as a by-product of oxidation amounts to 0.9 Gm. of water for each gram of solid food eaten.

The loss of water from the body is from three sources, i.e., vaporization, urination and in the stool. The skin and lungs dissipate body heat by vaporization. This vaporization process is continuous in that there is always some vapor in the breath and on the body surface. Even without visible sweating 25 per cent of the body heat is dissipated by water of vaporization. This insensible loss of body fluid amounts to 1,000 to 1,500 c.c. daily.[2–6] The remaining 75 per cent of body heat is normally

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dissipated by radiation, conduction and convection from the body surfaces. As visible sweating occurs, up to 100 per cent dissipation of body heat may take place by vaporization.

It is an interesting and important fact that vaporization is little affected by the amount of water available. Thus vaporization has priority over the kidneys on water available.

The kidneys, on the other hand, are markedly affected by the amount of water available. They excrete the waste material presented to them in water left after other processes have been cared for. Thus, there may be a large daily output of urine of a low specific gravity or vice versa. Especially in disease there is apt to be a deficiency in urine with a retention of some of the waste material in the blood stream with a resulting rise in the plasma non-protein nitrogen. In general, an adequate output of urine is indicative of an adequate fluid intake—i.e., *water balance.* However, as will be discussed later, this does not mean a proper salt metabolism. A small urine volume of high specific gravity usually means insufficient water intake, while excessive quantity of urine of a low specific gravity is indicative of the taking of excessive amounts of water.

TABLE 1

Kidneys	Maximum Concentrating Ability, Specific Gravity	Minimum Amount of Water Required to Excrete 35 Gm. of Waste Materials, c.c.
Normal.....	1.032–1.029	473
Diseased.....	1.028–1.025	595
	1.024–1.020	605
	1.019–1.015	850
	1.014–1.010	1,439

As an arbitrary standard 35 Gm. of waste material that must be excreted by the kidneys daily may be accepted for an average adult. Lashmet and Newburgh[7] determined the excretory capacity of the kidneys in health and disease. Table 1 (from Maddock and Coller,[5] based on Lashmet's and Newburgh's[7] work) indicates the minimum amount of urine of various specific gravities that must be excreted to remove 35 Gm. of waste material.

Persons with normal kidneys require a urinary output of only 500 c.c. if they concentrate to maximum capacity, while severely damaged kidneys with low concentrating capacity 1.010–1.014 require nearly 1,500 c.c. of urine to excrete the same amount of solids. The excretion of smaller quantities of urine for given concentrations will result in retention of waste materials and an elevation of the non-protein nitrogen of the blood.

In disease it is safer to assume that the kidneys have only a low concentrating ability and to strive for a minimum output of 1,500 c.c. of urine daily. While some believe 750 to 800 c.c. daily output of urine is adequate, if such quantities are accepted then frequent specific gravities of the urine should be determined and these should be 1.020 or above. Thus the hydration or dehydration of the patient is easily checked by determining the urinary output if the salt metabolism is normal. At the same time this daily observation will indicate whether enough solids are being removed.

Water lost in the normal stool is negligible. Since the function of the colon is to dehydrate the bowel content, an average of only 150 c.c. of water is lost daily by this route. This is interesting since from 7,000 to 10,000 c.c. of fluid are poured into the upper gastrointestinal tract daily. Obviously in cases of diarrhea the fluid loss by means of the stools may become alarming.

SALT (SODIUM CHLORIDE) METABOLISM

Water balance cannot be divorced from sodium chloride metabolism because the loss of this electrolyte is dependent on water to carry it away in solution. The normal daily salt requirement as given

by different authorities varies from 1 to 10 Gm.[8–11] We may compromise on 4.5 Gm. as the average daily requirement. This salt intake must be greatly increased in normal people working under abnormal conditions where there is excessive salt loss, as in stokers and steel furnace workers, etc.

Sherman[9] has estimated the total salt content of the body as 0.15 per cent of the body weight expressed as chlorides or 0.248 per cent of the body weight expressed as sodium chloride. The total sodium chloride content of a 60 kg. person would be 148.8 Gm.

Sodium chloride has two main body functions. As an electrolyte it aids in maintaining the acid-base balance of the body by its sodium and chloride ions. The other function is to aid in the distribution of body fluids and the regulation of their amounts by its influence on osmotic pressure.

In the role of acid-base balance, if the chloride ion is lost as in high intestinal obstruction, it is readily replaced by the bicarbonate ion.[10] Most significant is the fact that when the sodium ion is lost no other ion can adequately take its place. Therefore, in the loss of excessive amounts of sodium ion without the loss of chloride ion, chloride and bicarbonate ions are excreted in order to maintain the body acid-base balance. This results in a definite depletion of the total electrolyte content which in turn leads to a decrease of the total body water, or dehydration. Dehydration subsequent to loss of gastrointestinal secretion is due to the loss of sodium ions.

Salt balance in a healthy individual is of no practical significance. The chloride content of the blood expressed as sodium chloride (since pure chloride determination is not feasible) is 450 to 500 mg. per 100 c.c. of blood. Or if expressed as plasma sodium chloride, it is 550 to 630 mg. per cent. This blood chloride content in health does not change even if huge quantities of salt are ingested. The average person ingests daily from 8 to 12 Gm. of salt. About 2 per cent or 0.2 Gm. is excreted in the feces, 0.24 to 0.41 Gm. is lost through the skin in absence of sweating. Only 0.5 Gm. is normally lost through these channels, and the balance, after being used for body requirements, is excreted in the urine. The urine may contain up to 2 per cent salt. In cases of diarrhea, gastrointestinal loss, sweating, etc., the urine chlorides are decreased. The calculated body requirement varies from 1 Gm. daily[8] to 5 to 10 Gm.[11]

Excessive amounts of salt when ingested are removed in the urine or may cause diarrhea. The picture is different in disease[5] and the excessive sodium chloride is retained along with water, resulting in edema. This causes a hydremia and not a hyperchloremia.

A different situation occurs in cases of inadequate salt intake. When the salt ingested is below body requirements normal quantities of chlorides will be excreted in the urine for a few days then be reduced, but small amounts of chlorides will be excreted each day. A man fasting thirty-nine days excreted 20.3 Gm. of sodium chloride during this time, of which 50 per cent was lost in the first four days.[12] After several days of fasting the kidneys conserve salt by excreting less than 0.5 Gm. per day and if salt is lost elsewhere, as in an intestinal fistula, the urinary output falls below 0.1 Gm. per day.

Investigators have found that the loss of chlorides through normal and abnormal body fluid losses causes first a fall in the blood chlorides—hypochloremia—and second a depletion of the body chlorides in general. This is often followed by an alteration in the acid-base balance, and commonly there is an increase in the blood non-protein nitrogen as a later manifestation.[11]

If, instead of a loss of both the sodium and the chloride ions, there is a loss of one in excess over the other, then different blood findings occur. In the case of a loss of chloride ions—for example, in long

continued gastric aspiration—the chloride ion loss is compensated by an increase of the bicarbonate ion. Thus the water balance of the body is maintained if enough water is available. However, the reduced chloride content of the blood causes an alkalosis with an increased carbon dioxide combining power. In the absence of a sufficient amount of water the urinary output is reduced and the blood non-protein nitrogen is increased. If enough water is then supplied the total non-protein nitrogen falls, but not the carbon dioxide combining power of the blood.

The body has no substitute for the excessive loss of the sodium ion, and acidosis results, with a lowered blood carbon dioxide combining power. Body fluids are lost in excess and an increase in the blood non-protein nitrogen occurs. Giving water alone will not improve the dehydration but may even make it more severe. Only the replacement of the sodium ion will correct the condition.

The third possibility—equal loss of sodium and chloride ions—will not change the acid-base relationship, but dehydration will occur. Dehydration occurs in the body's effort to maintain the sodium concentration and the osmotic pressure at a constant level. If the salt loss and its accompanying water loss continue, eventually it is more important for the body to retain water than to maintain a constant sodium chloride level and then the blood chlorides fall. Bartlett[11] and his co-workers believe that the non-electrolyte urea is retained to maintain the osmotic pressure. The blood non-protein nitrogen is increased and the mere administration of water will not lower it significantly; salt is also necessary.

DEHYDRATION

Our first consideration is dehydration attendant on surgical operations. About 70 per cent of the fluid loss during an operation is due to vaporization. The remainder is due to blood loss, vomitus and urine secreted. The urinary secretion is small. Surgeons working on the pelvic organs for sixty to ninety minutes, or more, are not disturbed by the urinary bladder becoming filled. Preoperative dehydration of the patient can be avoided to a great extent by substituting parenteral administration of fluids if fluids cannot be taken freely by mouth.

In the operating room the loss through perspiration can be reduced greatly by using light drapes. Air conditioned and humidity controlled operating rooms are a great aid in this direction. The old "ether bed" added insult to injury by further sweating and dehydrating an already partially dehydrated patient. Except in cases of shock a comfortable bed with light covers is all that is needed.

The blood lost during surgical procedures is a factor in dehydration, depending upon the amount of blood lost. The figures in Table II were accurately determined for eleven cases.[5] The figures for the brain surgery represent the extremes of several cases.[13]

TABLE II
BLOOD LOSS DURING TYPICAL OPERATIONS

Operation	C.C. of Blood Lost
Partial gastric resection	274
Excision of thyroglossal cyst	174
Repair of inguinal hernia	54 and 147
Hemorrhoidectomy	8
Appendectomy	14
Excision retroperitoneal teratoma	546
Radical Mastectomy	1,272
Thyroidectomy	142 and 361
Repair of ventral hernia	306
Brain operations (White, Sweet and Hurwitt)	22 to 2,050

It is evident that the routine procedure of administering fluids either by hypodermoclysis or intravenously during the course of the operation is on a sound basis. In calculating the amount of fluids to be administered on the operative day it is wise to allow for 500 to 1,000 c.c. excessive loss during the course of the operation and immediately following. A custom I practice is to ignore the fluids given in the operating room in calculating the daily volume

of fluids to be given but not the salt given. Thus, if it is determined that the patient should receive 3,500 c.c. of fluid, this is given in addition to the fluids given in the operating room. However, if 1,000 c.c. of physiologic salt solution is given during the operation, while for the day 4,500 c.c. of fluid should be given, ordinarily the remaining 3,500 c.c. would be given as 5 per cent glucose in distilled water. The rôle of such fluids in preventing or lessening the degree of surgical shock also commends their use during the surgical procedure.

Postoperative Loss of Fluid. A smoothly convalescing patient will vaporize the same amount of fluid as will a normal person—1,000 to 1,500 c.c. in twenty-four hours. The presence of a fever will increase the amount of water lost by vaporization to as much as 2,500 c.c. A hyperthyroid state is characterized, among other things, by a warm moist skin and the water of vaporization is frequently 1,500 to 2,000 c.c. It must be remembered that this water of vaporization is lost before fluid is available to the kidneys.

Abnormal fluid losses may be great and if ignored will upset the water balance. Such abnormal losses may be from hemorrhage, vomitus or fluid aspirated from the upper gastrointestinal tract, diarrhea, biliary or intestinal fistula and excessive expectoration.

These fluid losses may be preoperative, postoperative or both. It is not uncommon to aspirate 500 to 1,000 c.c. or more of fluid from the stomach and duodenum when the indwelling catheter with negative pressure is used. In case of a biliary fistula similar amounts may be lost.[8] Severe diarrhea may cause enormous loss of fluids as well as of sodium ions. Excessive expectoration is rarely a cause of sufficient fluid loss to necessitate replacement. Large denuded areas may cause considerable fluid loss but usually the protein loss is more important.

We are now in a position to calculate the necessary water intake in order to maintain the water balance of the patient. (Table III.) It will be shown that such fluid must be properly balanced as to total salt content.

Table III

WATER NEEDED FOR EXCRETION PER 24 HOURS[5]

	C.c.
1. Insensible water loss	1,500 to 2,000
2. Urine	1,500
3. Abnormal loss (vomitus, etc.)	
	3,000 to 3,500

An average adult patient with no abnormal water loss requires a daily intake of approximately 3,500 c.c. of fluid. Where there is an abnormal water loss this should be quantitatively determined and an equal volume of water added to the fluid intake. If an indwelling catheter is in the stomach or duodenum and fluids are given by mouth, this quantity must be deducted from the total aspirated volume to arrive at a correct figure for the gastric-duodenal material aspirated.

To calculate the total water intake required to maintain the water balance in an adult with constant aspiration of the stomach and with the patient being permitted to drink water, it is necessary to add the net aspirated stomach contents to the basic requirements of 3,500 c.c. Thus, if 1,250 c.c. of material is aspirated from the stomach in twenty-four hours, but 750 c.c. of water has been drunk, then there is a net aspirated stomach content of 500 c.c. The patient should then receive 3,500 c.c. plus 500 c.c. or 4,000 c.c. of fluid in that twenty-four hours.

With many patients subjected to continued stomach aspiration preoperatively, it is well to remember that they may come to the operating room in a state of partial dehydration and even hypochloremia unless this fluid and electrolyte loss is replaced.

A rule worth repeating is that in general the simplest test of hydration of the patient is to measure the twenty-four hour urinary output. This should be 1,500 c.c.

WATER BALANCE OF THE DEHYDRATED PATIENT

If a patient is dehydrated before presenting himself for treatment or if he is permitted to become dehydrated, additional problems arise. The symptoms of dehydration are dry hot skin, parched tongue, slight fever, sunken eyes, scanty urine of high specific gravity. There may be albumin in the urine in the severe cases and the blood non-protein nitrogen is increased to as high as 40 mg. per 100 c.c.

When serious dehydration occurs, the water loss may amount to 6 per cent of the body weight.[5] A loss of about 10 per cent is incompatible with life. Table IV gives the amount of water equal to 6 per cent for various body weights.[5]

TABLE IV

Body Weight	6 Per Cent, C.c.
10 Kg. or 22 pounds	600
20 Kg. or 44 pounds	1,200
60 Kg. or 132 pounds	3,600
80 Kg. or 176 pounds	4,800

If a 176 pound patient is dehydrated he would theoretically need in the first twenty-four hours 4,800 c.c. plus 3,500 c.c., or 8,300 c.c. of fluid. A blood chloride determination is necessary to determine how much sodium chloride need be given (the method of calculation for this will be given later). Some workers prefer to hydrate such a patient over two or more days.

SIGNS AND SYMPTOMS OF HYPOCHLOREMIA

It is interesting that at times we attribute certain symptoms to the disease rather than to the reduced blood chlorides. The *symptoms* of hypochloremia are as follows:[11]

Group A: 1. Marked lethargy
2. Lassitude
3. Depression
4. Weakness
5. Fatigue

Group B: 1. Dulling sense of taste
2. Anorexia
3. Nausea

Group C: 1. Dulling of mentality
2. Drowsiness
3. Stupor
4. Coma

Group D: 1. Muscular twitching
2. Cramps

The less constant *signs* of hypochloremia are:

1. Dehydration—signs of which were listed before.
2. Alkalosis, with shallow slow respiration, and occasionally, tetany.
3. Acidosis may be present with deep rapid breathing.
4. Low pulse pressure may be present.
5. Shock.

Symptoms usually occur when the blood chlorides are 370 or below, but may occur at a much higher level. The diagnosis rests on the finding of low blood chlorides. Frequent blood chloride estimates are expensive and often impractical. In such cases the urinary excretion of sodium chloride can be determined. A roughly quantitative determination can be quickly made by the method suggested by Fantus.[14] The method is as follows: "Place 10 drops of urine in a test tube. Add one drop of a 1 to 5 potassium chromate solution. The fluid will now assume a distinctly yellow color. Add drop by drop, with the same dropper or one of the same caliber, a 2.9 per cent silver nitrate solution until a permanent and distinct color change to red-brown occurs (silver chromate). The number of drops required to produce the change of color expresses in grams the content of chloride per liter of urine." Fantus[14] believes that, except in emergencies, no patient should be sent to the operating room unless he has passed at least 1,500 c.c. of urine in the preceding twenty-four hours and unless this urine contains at least 0.5 per cent of chloride. Dehydrated patients with hypochloremia would not be operated on.

To prevent hypochloremia in surgical patients who cannot take fluids by mouth it is necessary to give a minimum of 500 c.c. of physiologic salt solution which

contains approximately 4.5 Gm. of sodium chloride. Unless contraindicated because of kidney damage we give 1,000 c.c. of physiologic salt or Ringer's solution. The abnormal fluid loss is replaced volume for volume with physiologic salt solution or Ringer's solution. This 1,000 c.c. of solution that is given routinely contains twice the normal daily requirement of sodium chloride and will compensate for the first 500 c.c. of abnormal fluid loss. In determining the gastric secretion, the amount of water drunk is deducted from the total aspirated volume to get the true amount of gastric fluid lost. The balance of the water must be given without the electrolyte.

If the patient has developed hypochloremia we are confronted with the problem of supplying enough sodium chloride to bring the blood chlorides up to normal. Orr[15] has suggested giving to such patients 250 to 500 c.c. of a 2.5 to 5 per cent solution of sodium chloride intravenously. This method will work but it is empirical, not based on a quantitative need, and the solution is definitely hypertonic. Bartlett[11] and his co-workers have evolved a formula based on the absolute requirement of salt of a patient of any weight. This formula makes use of Sherman's[9] determination of the normal body content of salt being 0.248 per cent of the body weight. The formula is:

$$\text{Gm.* salt needed} = \frac{450\text{-actual blood chlorides}}{450} \times 2.48 \times \text{kg. of body weight.}$$

From this formula a clinical rule simple in its application was formulated: For each 100 mg. that the whole blood chlorides need to be raised to reach the normal (450 mg.) the patient should be given 0.6 Gm. of sodium chloride per kg. or 0.25 Gm. per pound of body weight.†

In these determinations the lower normal level is used since it is not possible in many patients to elevate the blood chlorides above the lower limits of normal. If desired, the calculations may be made on higher values for normal.

According to the clinical rule, a 60 kg. person with a blood chloride of 350 mg. per cent requires 36 Gm. of salt to raise the blood chloride to a normal of 450.

$$\frac{100 \text{ mg. needed to raise to normal}}{100} \times 0.6 \times 60.0 = 36.0$$

If the exact formula were used instead of the clinical rule, it would be found that 33.0 Gm. of salt were needed. This discrepancy is insignificant clinically.

If only the dehydrated weight of the patient is known, the hydrated weight can be calculated as follows:

$$\text{Hydrated weight} = \frac{\text{Dehydrated weight}}{0.94}$$

The salt should be replaced as an isotonic solution, either physiologic salt or Ringer's solution. If physiologic salt is used the patient should receive

$$\frac{36.0}{8.5} = 4.2 \text{ liters.}$$

When it is not possible accurately to measure the abnormal body losses, management is difficult as too little or too much salt may be administered with a hypochloremia persisting in the former and edema occurring in the latter. Frequent blood chloride determinations may be made but the simpler and more rapid urine chloride determination of Fantus[14] is usually sufficient. While excessive salt secretion in the urine is usually indicative of excessive salt intake in disease, this is

* If chloride estimates are for plasma chlorides rather than blood chlorides, then the formula would be:

$$\text{Gm. salt needed} = \frac{560\text{-actual plasma chlorides}}{560} \times 2.48 \times \text{kg. body weight.}$$

† If plasma chlorides rather than blood chlorides are determined, the rule is: For each 100 mg. that the plasma chloride level needs to be raised to reach the normal, the patient should be given 0.5 Gm. per kg. or 0.2 Gm. per pound of body weight.

not necessarily true nor does the blood chloride content go above normal.

Certain complicating factors should be mentioned; the ideal management of these problems is still unsolved. In cases of edema, ascites, and hydrothorax a vicious circle often develops.[16,17] In an attempt to maintain the osmotic pressure of the transudate the blood chlorides are depleted. If large amounts of sodium chloride are given more fluid is retained.

METHODS OF ADMINISTRATION OF FLUID

1. By mouth (most desirable where feasible)
2. Proctoclysis
3. Retention enema
4. Subcutaneously
5. Intravenously

Needless to say, the patient able to take fluids by mouth and absorb the fluid from the gastrointestinal tract should receive fluids orally.

Proctoclysis or retention enemas of tap water or physiologic salt solution are the second most simple method of administration of fluids. However, there is a limit to the amount of fluid that can be absorbed from the colon. Such administration is distressing to many patients and should be reserved in certain cases for the administration of drugs, when the patient is unable to take drugs by mouth. Rectal administration of fluids and drugs is too often overlooked. Glucose, however, is not absorbed from the rectum.[18] McNealy[19] believes there is an advantage in administering by rectum the aspirated stomach and duodenal contents except in patients with carcinoma of the stomach.

The administration of fluids by hypodermoclysis is not a dangerous but too often a painful procedure. The needles should not be introduced near large vessels or nerves, nor should the fluid be introduced too near the joints. The axilla is the ideal location for the introduction of subcutaneous fluids because of the large amount of loose areolar tissue and the wealth of lymphatics. Probably the poorest location for hypodermoclysis needles is in or beneath the breasts. The widespread use of the breasts for such fluid administration has done more than any other single factor to bring a most valuable procedure into disrepute, especially in the estimation of the patient.

The use of large quantities of fluids subcutaneously calls for various sites for their introduction. The thighs offer a convenient location. The outer surface of the thigh is preferable to the inner side, although more painful, and absorption is less rapid. The inner aspect of the thigh is undesirable because of the universal contamination of this area and the frequent incidence of varicose veins. Fluids introduced about old varicose veins may cause an exacerbation of an old thrombophlebitis. Practically all sloughing of tissue secondary to hypodermoclysis occurs on the inner aspect of the thigh. An excellent practice in giving fluids by hypodermoclysis is to give 1 liter, remove the needles and, several hours later, reinsert the needles, giving a second liter. This procedure should be repeated until the desired quantity of fluid is administered. Isotonic glucose solution is more slowly absorbed from the subcutaneous tissues than is physiologic salt solution.

With the many refinements in the preparation of fluids, intravenous administration is the method of choice with many clinicians. It has the advantage of being less painful than hypodermoclysis. Chills and fever are rare occurrences following phleboclysis today, and if such do occur, the preparation of the rubber tubing should be carefully checked before blaming the accepted commercial preparations for intravenous administration. Occasionally such fluids are apparently at fault. Each flask should be carefully inspected for any change in color or gross foreign particles before administering it. The hazards of intravenous administration are avoidable by the exercise of reasonable care and skill. (1) The circulatory system may be overburdened by too rapid increase in the blood volume. The rate of flow of

intravenous fluids should be 250 to 500 c.c. per hour. In heart damage a lower figure is preferable. (2) General edema and edema of the lungs are secondary to overburdening the heart. (3) Thrombosis of the vein at the site of injection is easily avoided by removing the needle after completing the injection and frequently changing the veins chosen for injection. Varicose veins should be avoided as the site of injection because of the danger of lighting up a dormant thrombophlebitis, a serious complication in any illness. (4) Increase of the degree of dehydration of the patient by the use of hypertonic solution[20] is to be avoided. (5) Hassin[21] recently reported eight cases of nerve injuries subsequent to administration of fluids in the basilic and cephalic veins at the bend of the elbow. Care in introduction of the needle into the vein and avoiding spilling fluid outside of the vein will prevent such accidents. Veins on the dorsum of the hand are often useful for introduction of fluids.

FLUIDS THAT MAY BE ADMINISTERED

Water is the simplest fluid that may be administered by mouth and is the vehicle of all solutions taken into the body. As long as water can be given orally in adequate amounts we need not worry about other methods of administration. If necessary, sodium chloride or glucose may be added to the fluid given orally. Tap water or physiologic salt solution may be given by rectum. Too much water may be given, with a resulting water intoxication. Helwig[22,23] and his associates reported two cases of water intoxication associated with a lowering of the blood chlorides. One patient died and one recovered. The patient who recovered received 7,000 c.c. of tap water by proctoclysis in the first thirty-six hours after operation. At this stage the patient became unconscious and developed convulsive movements. The blood chlorides fell to 380 mg. per cent. Recovery followed administration of hypertonic sodium chloride solution and sodium chloride by mouth. There was an edema of the cerebral tissue in the patient who died.

It has been found[24] that approximately 70 per cent of the body consists of water which moves between intracellular and extracellular tissues to maintain an osmotic equilibrium between the intracellular and extracellular fluids. Sodium ions and chloride ions are the chief electrolytes of the extracellular fluids. Intracellular electrolytes are believed to be proteins, potassium and phosphates. Thus, as the salt is removed from the extracellular fluid, water passes into the cell to readjust the osmotic pressure with the extracellular fluid. As a result there is an edema and swelling of the cell, thus explaining the edema of the cerebral tissue in Helwig's fatal case of water intoxication.

Salt Solutions. Physiologic salt solution, Ringer's solution and Hartman's solution may all be considered under the classification of salt solutions. Physiologic salt solution is 0.85 per cent salt in distilled water. Ringer's solution contains slightly less sodium chloride, but in addition has calcium and potassium ions. In Hartman's solution there is sodium lactate as well as the constituents of Ringer's solution.

Physiologic salt solution is isotonic. If less than 0.85 per cent salt is contained in the solution, solution should be made isotonic by the addition of glucose.

Since physiologic saline solution contains 8.5 Gm. of sodium chloride per liter, a patient who receives 3,500 c.c. of such solution receives approximately 30 Gm. of sodium chloride or seven times the average daily requirement of 4.5 Gm. Samson-Wright[7] states that isotonic salt or similar solutions which are drunk are retained better than if given intravenously. Maddock and Coller[5] found that patients given 5 per cent glucose in physiologic salt or Ringer's solution retained water, less being retained if Ringer's solution was used. The work of Maddock and Coller demonstrated that salt metabolism in disease is not the same as in health and that

excessive quantities of salt in solution cause retention of water in the tissues. These workers found that patients receiving 5 per cent glucose in distilled water did not retain water in the tissues.

Glucose. A 5 per cent solution of glucose in distilled water in isotonic. This is our best method of giving water parenterally without the introduction of an electrolyte to upset the water balance. The water exchange when isotonic glucose solution is given is the same as if the fluids were given by mouth. Thus a patient requiring 3,500 c.c. of water in twenty-four hours would best receive this as 1,000 c.c. physiologic salt solution or Ringer's solution and 2,500 c.c. as 5 per cent glucose in distilled water.

Animals will tolerate up to 0.8 Gm. of glucose per kg. of body weight per hour when given intravenously without any trace of it in the urine.[8,25] When 0.9 to 2 Gm. of glucose per kg. of body weight are administered intravenously per hour some of the excess is eliminated in the urine. Translated into working terms, a 175 pound (80 kg.) man should tolerate up to 1,280 c.c. of 5 per cent glucose per hour or 640 c.c. of 10 per cent glucose per hour. Clinically it is found that when 5 per cent glucose is given at the rate of 300 to 500 c.c. per hour, 79 per cent of the patients will exhibit sugar in the urine and if 10 per cent glucose is given at the same rate, all will present sugar in the urine.[26] This may occur without an elevation of the blood sugar and the amount of sugar spilled is insignificant. In diabetics, as well as in non-diabetics, the sugar may be "covered" when desired by an adequate dose of insulin. More urine is secreted if 10 per cent glucose solution is given intravenously than if 5 per cent glucose is administered.

While an isotonic solution is physiologically sound there are occasions when hypertonic solutions are justifiable, but such cases are exceptions and not the rule. In patients with hyperthyroidism or liver damage 10 per cent glucose in distilled water is often the solution of choice. There are 200 calories per liter of 5 per cent glucose and 400 calories per liter of 10 per cent glucose solution, so where extra calories are needed 10 per cent glucose solution may be preferred.

Salt and Glucose Mixtures. It is in the use of mixtures or combinations of salt and glucose that we find the greatest number straying from physiologic principles. It is a practice of almost all commercial houses to prepare 5 per cent glucose in physiologic salt, Ringer's, or Hartman's solutions. In fact, many hospitals do not stock glucose in distilled water. Thus the first violation of physiology is to give every patient receiving such combination a hypertonic solution. While there is a large factor of safety, it is not necessarily always safe—if so, there is no reason for insisting on the salt solution, Ringer's and Hartman's solutions being isotonic.

Secondly, such combinations of salt and glucose lead to the intake of unnecessarily large quantities of salt, which cannot be removed by the sick patient and lead to edema. This error is due frequently to the assumption by the clinician that the glucose is dissolved in distilled water.

If it is desired to give a combination of salt and glucose, isotonic solutions of 0.425 per cent sodium chloride and 2.5 per cent glucose or variations of these constituents to maintain an isotonic solution should be given.

Blood transfusions and gum acacia solution should be reserved for shock, hemorrhage, severe anemia, and edema from protein loss. While there are recorded instances of protein shock and death from the use of gum acacia, many still consider it a valuable adjunct for maintaining the viscosity of the blood.

SUMMARY

1. Water metabolism and salt metabolism are the chief factors in water balance.

2. No water is available to the kidneys until the requirements of vaporization are satisfied.

3. Normal kidneys need a urinary output of only 500 c.c. if they concentrate to maximum capacity, while severely damaged kidneys with low concentrating capacity (1.010–1.014) require nearly 1,500 c.c. of urine to excrete the same amount of solids.

4. The average daily sodium chloride requirement of an adult is about 4.5 Gm.

5. The loss of the chloride ion causes an alkalosis. The loss of the sodium ion causes an acidosis.

6. The parenteral administration of fluids during the course of operations is a rational procedure. Such fluid should not be considered in calculating daily volume of fluid to be given but the salt content should be considered.

7. A simple test of hydration is to measure the twenty-four hour urinary output, which should be approximately 1,500 c.c.

8. In case of serious dehydration the water loss amounts to 6 per cent of the body weight.

9. The clinical rule of Bartlett for determining the amount of sodium chloride required to bring the blood chlorides to normal in cases of hypochloremia is satisfactory.

10. The indiscriminate use of hypotonic and hypertonic solutions is to be condemned. The routine use of 5 per cent glucose in physiologic salt solution is not physiologic in principle.

11. The average patient unable to take fluids by mouth should receive 500 to 1,000 c.c. of physiologic salt solution. The balance of the water requirement should be given as 5 per cent glucose in distilled water.

12. Patients with hyperthyroidism and liver damage may be given 10 per cent glucose in distilled water.

REFERENCES

1. Smith, H. J. The Physiology of the Kidney. New York, 1937 Oxford Press.
2. Coller, F. A. In Christopher's Surgery, page 1557. Philadelphia, 1936. W. B. Saunders.
3. Coller, F. A., and Maddock, W. G. Dehydration attendant on surgical operations. *J. A. M. A.*, 99: 875, 1932.
4. Coller, F. A., Dick, V. S., and Maddock, W. G. Maintenance of normal water exchange with intravenous fluids. *J. A. M. A.*, 107: 1522, 1937.
5. Maddock, W. G., and Coller, F. A. Water balance in surgery. *J. A. M. A.*, 108: 1, 1937.
6. Fuge, W. W., and Hogg, B. M. Insensible loss in surgical patients. *Ann. Surg.* 108: 1, 1938.
7. Lashmet, F. H., and Newburgh, L. H. A comparative study of excretion of water and solids by normal and abnormal kidneys. *J. Clin. Investigation*, 11: 1003, 1932.
8. Wright, Samson: Applied Physiology. London, 1935. Oxford University Press.
9. Sherman. Chemistry of Food and Nutrition, p. 242. 5th Ed. New York, 1937. Macmillan.
10. Gamble, J. L., and Ross, S. G. Dehydration following pyloric obstruction. *J. Clin. Investigation*, 1: 403, 1925.
11. Bartlett, R. M., Bingham, D. L. D., and Pedersen, S. Salt balance in surgical patients. *Surgery*, 4: 441, 1938.
12. Benedict, F. G. A Study of Prolonged Fasting. Washington, 1915. Carnegie Institute.
13. White, J. C., Sweet, W. H., and Hurwitt, E. S. Water balance in neurosurgical patients. *Ann. Surg.*, 107; 438, 1938.
14. Fantus, B. Fluids postoperatively. *J. A. M. A.*, 107: 14, 1936.
15. Orr, T. G. The therapeutic management of intestinal obstruction. *Surgery*, 1: 838, 1937.
16. Porges, O. Über Coma hypochloraemicum. *Klin. Wcbnscbr.*, 11: 186, 1932.
17. Mach, R. S. Les manifestations cliniques de l'hypochlorémie. *Rev. méd. de la suisse rom.*, 54: 829, 1934.
18. McNealy, R. W., and Willems, J. D. Absorption of glucose from the colon. *Surg., Gynec. & Obst.*, 49: 794, 1929.
19. McNealy, R. W. Personal communication.
20. Orr, T. G. Use and abuse of intravenous therapy in surgery. *J. Missouri State M. S.*, 34: 219, 1937.
21. Hassin, G. B. Nerve injuries caused by intravenous injections of dextrose. *J. A. M. A.*, 110: 948, 1938.
22. Helwig, F. C., Schutz, C. B., and Curry, D. E. Water intoxication. *J. A. M. A.*, 104: 1569, 1935.
23. Helwig, F. C., Schutz, C. B., and Kuhn, H. P. Water intoxication. *J. A. M. A.*, 110: 644, 1938.
24. Darrow, D. C., and Yannet, H. The changes in distribution of body water accompanying increase and decrease in extracellular electrolyte. *J. Clin. Investigation*, 14: 266, 1935.
25. Woodyatt, R. T., Sansum, W. D., and Wilder, R. M. Prolonged and accurately timed intravenous injections of sugar. *J. A. M. A.*, 65: 2067, 1915.
26. Winslow, S. B. Dextrose utilization in surgical patients. *Surgery*, 4: 867, 1938.

THE EBB AND FLOW OF DEATH

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LIFE does not move in the channels of old, and death patterns have accordingly changed. Montaigne remarked "Men do not usually die; they kill themselves." The degree to which changes have occurred in the causes of death, raises a question as to the validity of the statement of the philosopher.

Selwyn D. Collins[1] recently indicated the prevalence of particular forms of ailments at specific age levels. He directed attention to the fact that the major causes of death are not necessarily the most frequent causes of illness and hence, "The respiratory diseases are outstanding as causes of illness whether non-disabling or disabling; the degenerative diseases are more important as causes of death." He demonstrated, however, that these respiratory diseases constitute an overwhelming part of the sickness burden at every age and that the existence of digestive disorders is important throughout life. In the light of his statement it becomes important to make a somewhat thorough investigation of mortality, as it is recorded at varying age levels.

Death, as a frustration of personal continuity, obviously is a somatic reaction to environment in terms of constitutional alterations in vitality. The marked change in the modern expectation of life and the advancement of survival values, along with changing morbidity rates, represents, to a large extent, the modification of environmental conditions rather than any specific advance in constitutional capacities. Thus, if the aeroplane brings about an increasing fatality rate, there is necessarily a shift in rates for other accidents and diseases. With the decline of tuberculosis because of the alteration and control of environmental factors, there is an increased possibility for death to occur during the period of industrial activity from causes non-tuberculous in character. Similarly, it is obvious that with an increased recourse to surgery there is an additional possibility for an increased number of deaths following some ailments, operable or nonoperable, at an age earlier than such deaths might have occurred if no operation were performed. It is equally true that surgery has postponed many deaths to an age far beyond expectation. It is obvious that a considerable amount of shift in mortality rate is incidental to a very simple alteration in the environment. Insofar as infant mortality diminished under social attack, survival values to age 5 and above increased, as a result of which there were modifications of resistances which actually affected physical well being and altered, therefore, the percentile appearance of deaths from specific causes during the age 5 to 9 period. The development of first aid appliances, safety first teams, factory inspection and the like lowered mortality rates during the age period of industrial activity and thus enabled a larger group to advance into middle life and to die from causes especially lethal during the fifth and sixth decades.

Man's efforts to modify and better his environment have been constant during the ages but the advances of the past fifty years have been sharply precipitated by his scientific development. The Progress in Public Health charts, published in 1934 by the Metropolitan Life Insurance Company, reveal a remarkable contrast between the order of significance of the causes of death for 1900 and 1932. The following rates apply to the ten original states in the Death Registration Area and in the District of Columbia.

1900	Rate	1932	Rate
1. Tuberculosis	195	Diseases of the heart	280
2. Pneumonia	176	Cancer	123
3. Diarrhea and enteritis	140	Nephritis	90
4. Diseases of the heart	137	Pneumonia	82
5. Nephritis	89	Cerebral hemorrhage	82
6. Congenital malformations and diseases of early infancy	88	Accidents	70
7. Cerebral hemorrhage	77	Tuberculosis	59
8. Measles, scarlet fever, pertussis and diphtheria	76	Congenital malformations and diseases of early infancy	52
9. Accidents	72	Diabetes	28
10. Cancer	64	Suicide	19

It is notable that the total deaths from accidents had jumped from ninth place to the sixth place, while pneumonia fell from the second to the fourth place. Heart disease and cancer have risen to their higher positions because of many factors caught up in the changing age of our population, namely, the fact that a larger proportion now live to age 60, while there is a decreasing group of the population below age 25. One observes this definite rise in some causes of death incidental to the aging population levels and a falling off of other conditions once incidental to the earlier age period. If one notes the differences between the mortality rates for the two dates, one appreciates these distinctions very clearly. Thus, the rate of heart disease is more than doubled; cancer has almost doubled; nephritis is practically stationary but has risen in position; pneumonia has been reduced by half. One senses, therefore, shifts in values in terms of increasing, decreasing and stationary rates, which are not wholly caught up in the disease processes, per se, but are related to all the population changes in the general environment.

Sir Humphrey Rolleston[2] has suggested that the character of diseases such as scarlet fever, smallpox and typhus, has markedly changed and that this fact has been responsible for the modification of their fatality rates. He emphasizes, however, that environmental changes are as significant as those incidental to the biologic modifications of etiologic factors. People have changed in their internal environment as well as in their external environment; and the differential values of food, poverty, the stress and strain of life, are matters of biologic as well as of sociologic import. No one can deny that war altered the incidence of disease, increasing trench mouth, for example, while it diminished obesity; allowing an increase of rickets in the general population, and a decrease of venereal disease along at least one military sector. There can be no doubt that in an industrial age, electrification and motorization have heightened the speed of life, augmented the wear and tear of individuals, and produced a profound effect upon blood pressure, cardiovascular-renal reactions, and the activities of the sympathetic nervous system. One would expect, therefore, the evident increased fatality rate from diseases of the circulatory and renal systems as well as the manifest rise in the mortality from diabetes and conditions secondary to arteriosclerosis.

It is obvious that fatalities must be altered as the result of human accomplishment. A group in the community who might have died from the now obsolescent typhoid fever, cholera, typhus, gastro-entero-colitis, relapsing fever and malaria, now must die from some other ailment during the same relative age period, or succumb to some other disease at a later age. It is also quite apparent that many diseases appear to have had their natural history altered; thus, syphilis appears to be less malignant; acute rheumatism and gout are rarer; cirrhosis of the liver seemingly has declined; chlorosis has practically disappeared. On the other hand, numerous other diseases have been conditioned into existence, especially the large group known as occupational diseases, whether made manifest in the form of dermatitis, neuroses or psychoses.

Not alone have diseases undergone transformation, but one is able to note to a considerable degree the varying effects that they have had at different age levels. It is difficult to secure long range figures in the United States, but fortunately a few figures of Boston give some slight but impressive information concerning the average death rates from 1830 to 1930.[3] (Table 1.)

TABLE 1
AVERAGE DEATH RATES IN BOSTON 1830–1930

Age Period	Average Death Rate	
	1826–1835	1929–1931
All ages	21.3	14.4
Under 5 years	64.8	25.6
5–9 years	8.5	3.1
20–29 years	10.7	3.9
40–49 years	22.7	10.7
70–79 years	91.2	93.6

The death rate dropped approximately one third at all ages, and one notes especially the fall of more than 50 per cent under age 5, and at 40 to 49 years approximately 63 per cent, with a tendency to increase only above age 70. It is impossible to compare the causes of death of these two periods because of the differences in nomenclature and the altered adequacy of diagnosis incidental to advances in medical knowledge, including, of course, a greater familiarity with microscopic techniques.

Topographic variations in death rates are notable with the lower rate in the Mississippi Valley south from the Dakotas, and the higher mortality rates on the Eastern coast with its industrial centers. There are also differences due to population characteristics of age, sex, marital status, prevalent nationalities, economic forces and recency of settlement, etc., which enter into all tables of mortality.

Farr has stated: "There is relation betwixt death and sickness; and to every death from every cause, there is an average number of attacks of sickness, and a specific number of persons incapacitated for work." I am not concerned for the present with the ratio between death and specific sickness, but recognize that a higher fatality rate does not necessarily bear a continuing direct ratio to the incidence rate. Thus certain diseases in one epidemic have a high mortality rate, and in another a low one, as has been amply demonstrated in epidemics of infantile paralysis and encephalitis, scarlet fever and diphtheria. I desire to present here primarily an analysis of death. It is difficult to select a figure that will adequately make evident the marked hazards from various disease for different age groups.

If one takes the deaths in the city of New York[4] one finds that the average number of deaths from 1900 to 1909 was 73,190, with a rate of 18.32, while in 1929 the total number of deaths was 77,482, and the rate was 11.34. If one considers only the absolute number of deaths one notes a slight increase, although the death rates reveal a remarkable fall. The actual differences are not made clear because the population figures are not given. The ratio of total deaths is 1 to 1.06, indicating that the absolute increase of deaths is exceedingly small; the ratio of death rates, showing a marked decrease, is 1 to 0.62. These figures demonstrate at once, and sharply, the ratio differences.

It is necessary to sense our achievements and accomplishments in affecting mortality, not in blanket rates, but in relative rates at varying ages, and in terms of the relation of these population groups at the varying ages to their own increase or decrease as well as in terms of their relation to the total population increase. It is a significant fact that the percentage of the United States population in the Death Registration Area rose from 41 per cent in 1908 to 97 per cent in 1929, while the death rate in the Death Registration Area during these years fell from 14 to 12. Perhaps it would be more expressive to show the relations between population increase and absolute mortalities than merely to dwell in

terms of mortality rates which do not indicate exactly what has occurred.

Life expectancy during the Middle Ages approximated 21 years; in the United States in 1915 it was 48 years, in 1925, 55 years, in 1926, 57.74 years, and it has since increased to 60 years. These figures arose from an alteration in the incidence of diseases as well as their fatality rates and bear witness to the fact that the greatest improvements have attended the early years of life. Admittedly a change in any death rate, taken as a criterion of social improvement, carries with it many implications. Thus it suggests changes in nomenclature, alterations in diagnosis, increased knowledge concerning the nature of morbidity and heightened therapeutic skill, etc. On the other hand, it may indicate variations in susceptibility to specific illnesses or, indeed, variations in the numbers of people in various age groups exposed to or afflicted by the specific disease.

It is patent that gross measures of mortality at all ages cover up the rise or fall in the mortality at any special age level. Thus, the average aggregate number of deaths from a specific disease covers up the number which occur at any particular age, just as a total mortality rate gives no information concerning the mortality rate from individual diseases. There might be a falling gross mortality rate, with a definite increase in the mortality for some particular age periods. There may be falling gross mortality rates from various diseases which, nonetheless, include an increase of their mortality rates during later decades of life. It is apparent that with the case incidence and fatality rates constant, the number of people dying from any one disease would vary directly with the population. But neither the incidence rate, the fatality rate nor the population are constant. Hence it becomes helpful to contrast the ratio of the total population or a specific age population with the ratio of the number of absolute deaths during any two periods of time. I shall illustrate this without reference to specific diseases.

Dr. Walter F. Willcox[5] shows the age distribution of deaths in the Death Registration section of the United States for 1900 and 1930. The deaths per 100,000 population for varying age groups are indicated in Table II.

Table II
Deaths per 100,000 population

	1900	1930
Under 1 year	20,793	9,275
5 to 9 years	2,702	1,541
25 to 29 years	4,249	2,646
45 to 49 years	3,922	5,426
75 to 79 years	5,402	9,886

This tabulation gives a sense of values and shows the decrease under one year and the increase above age 45 years, but the decrease is illustrated far better by the following table of ratios, which, on the basis of employing the 1900 figures show the ratios to 1930 data to be as follows:

Under 1 year = 1 to 0.45
5– 9 years = 1 to 0.57
25–29 years = 1 to 0.62
45–49 years = 1 to 1.39
75–79 years = 1 to 1.83

This presents more adequately the definite rise above age 45 years, and the marked decrease under age 10, which would be swallowed up in the figures for the general mortality rate. These ratios, however, refer only to death rates and do not show the relation of these rates or ratios to the actual populations of the age groups thus enumerated, any more than they suggest the diseases responsible for the gross figures.

The marked changes in death rates for specific diseases are illustrated, for example, by data from New York State. It is clear that the differential mortality rates are wholly concealed in total death rates of the two years compared. (Table III.)

Herein it is evident that during the twenty-one year period prior to the depression era there had been a modification in the mortality from many diseases. The

TABLE III
DEATH AND DEATH RATES, NEW YORK STATE[6]

Diseases	1908	1929
Typhoid and paratyphoid	15.6	1.3
Diphtheria	28.1	5.3
Pulmonary tuberculosis	164.1	68.5
Cancer	74.7	121.8
Diabetes	14.1	26.2
Cerebral hemorrhage, embolism, thrombosis	85.1	59.7
Diseases of the heart	164.0	293.3
Diseases of arteries	24.1	53.1
Pneumonias	169.2	124.1
Diarrhea and enteritis under 2 years	23.8	3.5
Appendicitis	10.2	15.2
Acute and chronic nephritis	112.6	76.2
Accidents and other violence	79.4	81.2

three leading causes of death for 1908 were pneumonia, pulmonary tuberculosis, and diseases of the heart, whereas in 1929 diseases of the heart led, followed by pneumonia and cancer. The falling off of typhoid, paratyphoid, diphtheria and tuberculosis is no more striking than the increase in diabetes, diseases of the arteries and appendicitis.[7]

Analyzing the mortality rates in terms of months reveals some rather significant data. The general population does not alter much during the months nor are there notable age variations, and yet the fact that some diseases are far more costly in human lives during some months than in others suggests the existence of some mixed factors which call for investigation. Thus in New York State, in 1932, the total number of deaths was 146, 639. The first three months in order of fatality significance were March 16, 202; December 14, 075; and April 13, 717. If one plots the various diseases in terms of their fatality prevalence in any one month, one finds that March is attended by the highest mortality rate for pulmonary tuberculosis, diabetes, diseases of the heart, total pneumonia, appendicitis, hernia and intestinal obstruction. This is an unusual mixture of fatal conditions. The month of December was the month of maximum deaths from cancer, cerebral hemorrhage, embolism and thrombosis. April was not a month of maximum deaths from any disease. February and July lacked superiority in any fatality. Perhaps it would also be of advantage to observe these various diseases in the descending mensal order of their fatality—as typhoid and paratyphoid in August, October, September. (Table IV.)

TABLE IV

Diseases	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.
Typhoid, paratyphoid	..	..	..	..	..	..	..	1	3	2		
Pulmonary tuberculosis and acute miliary tuberculosis	..	..	1	3	2							
Cancer	2	..	..	..	3	..	..	..	..	..	..	1
Diabetes	..	..	1	3	..	..	..	..	..	..	..	2
Cerebral hemorrhage, embolism, thrombosis	..	..	3	2	..	..	..	..	..	..	..	1
Diseases of heart	..	..	1	..	3	..	..	..	..	..	..	2
Total pneumonia	..	..	1	3	..	..	..	..	..	..	..	2
Diarrhea and enteritis under 2 years	..	..	..	3	..	..	..	..	1	2		
Appendicitis	..	..	1	3	..	..	..	2				
Hernia and intestinal obstruction	2	..	1	..	..	3						
Premature births	3	..	..	..	1	2						
Automobile accidents	..	..	..	..	..	..	..	..	2	1	2	

It is worthwhile noting that while these figures indicate very definitely that the month of March is the most hazardous month in the year so far as fatality from these particular diseases is concerned, there are some diseases which had their highest mortality in May, August, September, October and December. It is important

to note that while these diseases have been rated as first, second and third in series, for some diseases there was comparatively little absolute variation. Typhoid fever, for example, ranged during the months only from 3 to 23; tuberculosis from 523 to 734; cancer from 1,256 to 1,450; diabetes from 245 to 397; cerebral hemorrhage, embolism and thrombosis from 457 to 683, the variation among the three highest months being only from 637 to 683; diseases of the heart from 2,559 to 4,239; pneumonia from 435 to 2,317; diarrhea and enteritis under 2 years, from 50 to 101; appendicitis from 122 to 202, the first three places varying only from 183 to 202. Hernia and intestinal obstruction varied from 81 to 131, the highest three months varying from 111 to 131; premature Births from 217 to 297, the highest three months varying from 273 to 297; automobile accidents from 194 to 284. Thus it appears that for certain diseases the death distribution through the months of the year 1932 was very nearly equal, indicating that the environmental factor probably was reasonably constant. These figures relate only to New York State for the one year.

I now offer a slightly different set of diseases (Table v) for the Registration Area of the United States for the year 1929, which demonstrates another set of facts for the country as a whole. Thus the figures 1, 2 and 3 indicate the months of greatest mortality significance and 10, 11 and 12, the months of lowest mortality, not in terms of rates but in terms of absolute mortality figures during the year. This chart indicates that for the country as a whole during 1929 the month of January was far more fatal than any other month during the year. Thus the environmental factors of the New York State in 1932 find no implication of a duplication throughout the United States during 1929. It is rather significant that the month of May is the only month without an allocation of a disease in the category of the very severe or of the very light fatality rates.

TABLE V

Disease	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.
All diseases in the Registration Area	1	3	2	..	..	..	10	11	12			
Typhoid, paratyphoid	10	11	12	..	..	..	3	1	2			
Malaria	10	12	11	..	..	..	..	1	2	3		
Pulmonary tuberculosis	1	..	2	3	..	..	..	..	12	10	11	
Diabetes	1	..	3	..	..	11	10	12	..	..	..	2
Lethargic encaphalitis	1	2	3	..	..	..	11	10	12	..	..	2
Cerebral hemorrhage and softening	1	..	3	..	..	..	10	11	12	..	..	3
Other diseases of the heart	1	2	..	..	..	..	10	11	12			
Bronchopneumonia	1	2	3	..	..	..	12	11	10			
Pneumonia	1	2	3	..	..	..	12	11	10			
Whooping cough	1	..	..	..	..	..	3	2	..	10	12	11
Influenza	1	2	3	..	..	..	11	12	10			
Diarrhea and enteritis under 2 years	11	12	..	..	..	..	3	2	1	..	..	10
Diarrhea and enteritis 2 years and over	11	12	..	10	..	..	3	1	2			
Automobile accidents	11	12	10	..	..	..	..	..	1	2	3	
Nephritis	1	..	3	..	..	..	10	12	11	..	..	2

If these figures were broken down into the absolute number of deaths from the specific diseases occurring in different topographic areas there would be definite variations of mensal distributions because of climatic differences and differential social phenomena.

Types of lethal diseases may be classified as organic and functional. Among the organic are those whose origins are: (a) infectious; (b) metabolic; (c) traumatic; (d) neoplastic; (e) congenital, hereditary and acquired; (f) toxemic.

The prevalence of deaths from infectious diseases such as typhoid and malaria is more dependent upon social factors than that of deaths from measles and scarlet fever, while those from tuberculosis, gonorrhea and syphilis are bound up in personal and social relationships.

The metabolic mortality is related mainly to nutritional problems, save as some are caught up in endocrine diseases. The traumatic group includes deaths from accidental slipping, which may be deemed personal, as well as from accidents incidental to automobiles and other transportational facilities, which involve personal and social relations. So also the deaths arising out of war or some phases of surgery certainly appear to have more social than personal origins. The neoplastic group for the most part may be considered as growing from personal rather than social factors, so far as we know at the present time, despite the suggested high incidence of neoplasms among chimney sweeps, etc. The congenital acquired disorders, so far as they are due to birth injuries may be said to be in part social, though the majority of congenital causes of death involving hereditary factors can scarcely be classified as either personal or social. Toxemic diseases vary from those which are due to industrial poisonings, which largely have their origin in social inadequacy, to deaths from alcohol in which the personal factor is primary, although, of course, alcoholism has its social aspects.

The functional diseases such as neurosis and psychosis, though not regarded as direct causes of death, save suicide, are related to some other diseases of real organic quality such as gastric and duodenal ulcers, and even indeed, to some arteriosclerotic changes, to which I shall refer later. Possibly I should include a separate category of degenerative diseases, which are thoroughly bound up in personal and social factors, and which affect the basic systems of man's organization, such as the heart, the blood vessels and the kidneys. The diseases of the cardiovascular-renal systems represent in large measure the personal reactions of the individual to social living. They appear to be a consequential expression of man's natural deterioration to the final lesion causing death, for the fact remains that death constitutes a part as well as the end of life.

I have already implied that many diseases have changed in their characteristics. Yellow fever is obsolescent; malaria has diminished; tuberculosis has decreased extensively especially in this country; diphtheria is controllable; typhoid fever has almost disappeared; summer diarrheas are largely traditions. Qualitative changes have appeared in connection with measles, scarlet fever and possibly whooping cough, incidental to factors related to some diminished virulence of the infective organism or virus or to a heightened immunity on the part of a younger generation. On the other hand certain diseases are increasing, such as lethargic encephalitis, poliomyelitis, diabetes, appendicitis, biliary calculus, and the like, in addition to many new forms of poisonings incidental to advancing industrial development. Hence, while plumbism and phossy jaw have disappeared, the incidence of radium poisoning, silicosis, benzol poisoning and the like, has increased.

Mortality as well as morbidity merits consideration as a reaction to living. Perhaps the age distribution of mortality suggests the resistance coefficients of different systems of the organism at different periods of life. The prolongation of life depends upon a harmonious interaction of organ systems and the resistance capacities of the organism as a whole. Certainly longevity, while it may contain a very definite factor from inheritance, also involves numerous elements bound up in what may be termed the art of active living. To live to a ripe old age certainly indicates an organic capacity for adaptation to the stresses of the life to which the individual was subjected. Noting the main causes of death which are still increasing in their ratio at ages 75 to 79, it follows that

in order to achieve a ripe old age, one must escape not only mortality in infancy, childhood and active maturity but such special ailments of middle age as appendicitis, diabetes, intestinal obstruction, biliary calculus and gastric ulcer. Deaths such as those due to cancer and angina pectoris have less significance upon the extension of life than the ability to escape death from an automobile injury. It is strikingly true that the most significant factors in promoting longevity are bound up with the welfare of the gastrointestinal and the circulatory systems.

In ordinary discussions of the prolongation of average life, a tremendous amount of emphasis is bestowed upon heredity, racial characteristics, diet and national tastes, habits of work and play, climate, economics, social position and vocational status, as well as exposure to specific forms of disease. When, however, mortality rates are consulted, it is more or less surprising to find that the most significant causes of death reveal little direct relation to any of these items, at least not upon superficial examination. The increasing ratios of some of the diseases cannot be analyzed in terms of all of these potential factors. It is challenging to find the large amount of mortality due to diarrhea, gastric ulcer, appendicitis and diseases of the gallbladder, as representative of the maladjustments of the gastrointestinal system. Questions arise from the fact that an organic disease of the heart, such as endocarditis, which apparently has diminished in rate among children and adults up to 45, is increasing at the later ages. Thus, endocarditis had risen as cause of death from third place in 1900 to first place in 1935.

The pneumonias among the respiratory diseases are important lethal agents, but far more significant is the fact that tuberculosis fell from first rank in 1900 to the sixth rank in 1929. The diseases of the excretory system are by no means as prominent as they were, while the neurologic disorders, although tremendously effective in disorganizing living, seemingly play but a small part in the general scheme of the direct causes of death. Functional disorders and organic diseases of neurologic significance are credited with limited serious responsibility for fatality, except as encephalitis and poliomyelitis in epidemic form take their toll. Even in old age, while neurologic and psychiatric disorders may be active accompaniments, they are not primarily registered as significant causes of mortality.

Raymond Pearl regards old age as being more dependent upon an inherited factor than upon any elements derived from the environment. While this may be true, the evidence for it is by no means certain. The inheritance element depends upon its social struggle. No one can question the fact that social organization has set up environmental conditions predisposing to survival through the control of water supplies, food supplies, food handlers; through the development of efficient health departments, with greater attention to prophylaxis and improved conditions of housing; through public provision for inoculations and vaccines; through great advances in education in personal hygiene, in dietetics, in child welfare, etc. It cannot be gainsaid that the limitation of typhoid and paratyphoid and typhus fevers, the control of diphtheria, yellow fever, bubonic plague and malaria, and the decrease of infant mortality have not been primarily dependent upon personal effort or personal inheritances. The most definite and effective reductions in mortality have been attained through social organization and the coöperative efforts of private agencies and public Departments of Health, Education, Sanitation, and the like, whether at the federal, state, municipal or county level.

It is quite evident that the level of personal hygiene in this country is probably higher than in most parts of the world, considering all levels of population. This does not mean that there is not ample reason for providing opportunities for better personal hygiene, because personal

hygiene of the highest type such as depend upon bathing, garbage and sewage disposal, adequate sunlight and fresh air, can be accomplished only in the light of social provisions. It is evident that heightened social efforts to promote healthful habits of living have involved a greater appreciation of foods, food constituents and food values, the promotion of sane eating habits; the development of recreational programs along with the establishment of adequate hours of rest; the recognition and removal of vocational hazards; a willingness to undertake certain personal and group responsibilities for protection against industrial disease and accidents; and indeed, even a greater appreciation of the value of mental hygiene in terms of diversion, occupation and the promotion of ego values.

This increased personal hygiene has played its part in the general welfare of people. The question arises, however, as to the extent to which this personal hygiene has been effective in diminishing mortality rates from causes which would appear to have some direct relation to the individual's mode of life. This question will receive definite consideration when I discuss specific diseases taking their toll from various age groups. It is obvious, however, that instruction in personal hygiene is less effective than the social application of a principle that compels personal hygiene without choice. If all milk is pasteurized by legal demand, the hazard of milk-borne diseases almost disappears, while the personal knowledge of the value of pasteurization may be disregarded in actual living. Personal hygiene should be a natural by-product of socialized living.

The rise and fall of mortality rates has its greatest significance in terms of the total population of specific groups. It is especially important to note the rise and fall of mortality rates with relation to the rise and fall of population groups at special ages. If, during some unit of time, the total population of any age group doubled and the total number of deaths doubled, the gross change would be the same but the nature of the combination of diseases entering into the total deaths would be concealed. If one notes the total deaths from any single disease, and plots their rise or fall with reference to the total population for any time sequences, it is possible to observe any marked variations that may occur. This becomes more valuable if the figures for the disease are compared with the numbers of people included in different age groups. It is obvious, therefore, that a study of the changes in the mortality rates at varying ages offers a sensitive approach to the consideration of dominant disease factors, since it demonstrates what has actually happened at particular age sections along the general highway of life. The information thus made available is superior to that derived from a consideration of the total mortality, or a disease rate, which, because it covers up internal variations, is not a refined index of how any specific disease affects our population.

Admittedly, an increasing population would serve as one explanation for a rise in the gross mortality from certain diseases, but this does not explain a rise in the mortality rates. Thus, an increased number of deaths at *all ages* from appendicitis, is understandable without increase in population, but not the higher mortality rate from appendicitis.

I shall present some problems of mortality which appear to indicate that the greatest drop in mortality rates has occurred where social action has been most effective, and that equal accomplishments have not been secured in a large number of diseases where cure has depended upon personally controlled living. Several death causes, now rising disproportionately to the increase of the population, generally, and in terms of specific age groups, should perhaps be placed in the category of diseases of concern from the standpoint of public health. Cannot special action postpone many deaths now apparently inevitable, at ages far too young?

In an analysis of this problem, I have employed the United States population figures 1910 and 1930, even though I shall discuss deaths for the years 1908 and 1929. By rejecting the years from 1930 to 1936, I escape the questions raised by the unusual economic forces of the depression. All data concerning population at the various ages have been secured directly from the United States Bureau of Census. I have not employed the comparative figures because, in 1910 only twenty-one states and the District of Columbia were listed therein, whereas in 1930 all states were included, except Texas and South Dakota. I cannot use the figures for the Birth Registration Area as this was not established until 1915, and there were practically no figures for 1910. It therefore seems more reasonable to employ the figures for the United States census as a whole. The value becomes more obvious, if one considers the total population. For example, in 1908, the total population in the Death Registration Area was 46,790,000, which constituted 52.5 per cent of the total United States population. The death rates in that area for the year 1908 was 14.8 per cent. In 1929, the population in the Death Registration Area was 116,310,000, approximately 95.7 per cent of the total United States population, and in this area the death rate was 11.9 per cent. The total deaths in 1908 were 691,574; in 1929 they had risen to 1,378,675. It is patent that while the death rate dropped from 14.8 per cent to 11.9 per cent (ratio 1 to 0.80) the actual number of deaths rose in the ratio of 1 to 2, while the population ratio had risen to 1 to 2.5. The relation of the total death ratio to the population ratio gives a clearer picture of what happened than the death rates or their ratio. I have completely ignored the change in the urban-rural percentages in the United States, which underwent marked changes between 1908 and 1929.

It appeared wise to choose for special consideration certain age periods which have definite influence in our general welfare. Hence, I have selected deaths under 1 year (prenatal, natal and infancy); deaths 5 to 9 years (childhood), 25 to 29 years (maturity); 45 to 49 years (middle age); 75 to 79 years (old age). I have utilized the absolute figures and have established various ratios of the total number of deaths from specific causes in 1929 with relation to 1908 as a base; also as the occasion required, I have set forth the various population ratios for purpose of comparison. By employing this system of ratios one at once is enabled to note which particular diseases have increased above the general increase for any or all age levels; whether the increase or decrease of mortality deviates from the population increase or decrease of any age group; and which diseases in terms of absolute numbers dying show a differential mortality at specific age levels as opposed to the total number at all ages. (Table VI.)

Table VI
Ratios 1908–1929

Deaths	Under 1 Year	5–9 Years	25–29 Years	45–49 Years	75–79 Years
Total deaths	1:1.07	1:1.6	1:1.5	1:2.2	1:3
Malformations	1:1.81				
Congenital heart disease	1:1.65				
Childbirth	.....	.....	1:1.9		
Respiratory system	.....	.....	1:1.6		
Disease of digestive system	1:2.4	1:2	1:1.8		
Appendicitis	1:5.2	1:3.2	1:3.2	1:4	1:6
Diabetes	1:1.66	1:1.7	1:1.7	1:2.5	1:5.5
Cancer	1:1.9	1:4	1:2.3	1:4	1:5
Accidental gunshot	.....	1:3.1	1:2.3		
Encephalitis	.....	1:4	1:9.3	1:4.6	
Lethargic encaphalitis	.....	1:2.4	1:9.8	1:4.6	
Automobile accidents	.....	1:45	1:92	1:83.5	1:90.5
Biliary calculus	.....	.....	1:3.7	1:3.2	1:3
Gastric ulcer	.....	.....	1:2	1:4.3	1:3.8
Intestinal obstruction	.....	.....	1:2.2	1:2.3	
Circulatory system	.....	.....	1:1.7	1:3	1:4
Angina pectoris	.....	.....	1:2.1	1:5.7	1:6.7

Table VI presents the ratios for the twenty-one year period from 1908 to 1929. It indicates that the total number of deaths under one year of age had risen only slightly, i.e., in the ratio of 1 to 1.07, while certain definite conditions revealed mortality ratios excessive in comparison with this ratio. Malformations, for example, were above the total ratio levels for the age period, as were deaths from congenital

heart disease. Most noticeable, however, are the heightened ratios of deaths from diseases of the digestive system, appendicitis, diabetes and even cancer.

I am offering ratios and it matters little that the absolute figures for these various diseases may have varied from units to a hundred thousand; the ratios carry a constant value. Thus, if there had been five deaths from a particular disease in 1908, and there were ten deaths in 1929, the ratio would be 1 to 2, just as would be the case if there had been ten thousand dying in 1908 and twenty thousand in 1929. The emphasis rests upon the ratio of absolute numbers rather than in terms of specific mortality rates. Table VI, therefore, demonstrates the actual relative increase of each listed disease, without reference to population rates or ratios. It shows that there was a marked increase in the total number of deaths at ages 45 to 49 and 75 to 79. Further, it makes clear that even within these age groups there are certain specific diseases which stand out markedly with ratios of mortality far above the average mortality ratio for the age group. I shall make the meaning of this more apparent by a contrast between the special disease ratios and the population ratios, as well as the total death ratios for these age periods.

For the moment it suffices to call attention to the fact that appendicitis and diabetes are responsible for higher ratios of death than those representing total deaths for each one of the five periods under consideration, while deaths from automobiles are responsible for greatly increasing mortality ratios in all groups save the first. Even more thought must be given to such outstanding rises in mortality ratios as are presented by biliary calculus, gastric ulcer and intestinal obstruction, especially since these appear to be as significant in the age group 25 to 29 years as the ratios of death from diseases of the circulatory system or angina pectoris.

While some diseases show up especially in the mortality of special age groups, a few diseases markedly evidence an increased mortality throughout the whole range of life from infancy to old age. Appendicitis, diabetes and cancer are outstanding, as are biliary calculus, gastric ulcer, diseases of the circulatory system and angina pectoris, whose havoc becomes manifest from the age of maturity onward through old age. While ratios of deaths from automobiles cover all periods save the first, the tremendous increase of the figures is due mainly to the fact that automobiles were less prevalent in 1908 than in 1929 and hence the possible exposure to this cause of death was much less.

The chart as a whole, clearly reveals some trends of mortality above those implied in the general mortality trends of the age. This is perhaps more evident in Table VII which, however, is based only upon the deaths of New York City males for a three year average and reflects the percentage of mortality taken by various diseases at each age. This table shows that pneumonia as a single cause is highly operative at each age level. It gives, however, no indication as to whether the pneumonia fatality or any other mortality is higher than the general mortality level of all diseases for the age period. It indicates the five chief causes of death in certain large age groups based upon the average number of deaths of males per year in New York City, 1932–1933–1934. The chart shows the percentage of total deaths in each one of the age groups, stressing only the five main causes of death.[1]

Table VII is wholly inadequate for giving the type of information available in Table VI, as it affords no evidence of the dynamics of a disease in terms of its rise or fall with relation to the general mortality ratio at any age, or with relation to the population changes or aught else.

It is generally stated that infant mortality rates have greatly diminished, but certainly Table VI indicates that some marked problems in infant mortality continue to demand attention. A rising mortality at later ages is to be expected, but as will be

Table VII

NEW YORK CITY MALES 1932–1933–1934: PERCENTAGES OF TOTAL DEATHS

	Under 1 Year	5–8 Years	25–29 Years	45–54 Years	65 Years and Over
Premature births	28				
Malformations and debility	10				
Other diseases of early infancy	16				
Pneumonia	21	11	9	9	8
Diarrhea	9				
All other causes	16	40	35	35	19
Accidents	..	27	16	9	
Heart disease	..	9			
Ear and mastoid	..	7			
Appendicitis	..	6			
Tuberculosis	..	..	23		
Circulatory diseases	..	..	11	31	49
Suicide	..	..	6		
Cancer	..	..	..	12	14
Nephritis	..	..	..	4	7
Diabetes	..	..	..	..	3
Puerperal diseases (for female group)	..	..	15		

found later, the rises in mortality are not dependent wholly upon increases in population. It is helpful to consider the general mortality ratios of different age groups during 1908 and 1929 and then to determine which disease ratios rose above them, recognizing, of course, that the total ratios as averages are affected by the fact that some disease ratios are high and others are low. The contrast between the average mortality ratio and the special higher mortality ratios indicates where certain problems of public health await solution.

Admittedly the greatest mortality rate should occur above age 60. Under ordinary conditions one would expect the deaths of the aged to be due largely to such degenerative diseases as arteriosclerosis, chronic nephritis, myocarditis, cerebral hemorrhage and the like. Table VI, however, indicates that certain causes of fatality are operative which are not peculiar to old age. These have risen disproportionately beyond the actual increase of total deaths to such an extent as to indicate that they merit consideration for reasons not inherent in the normal aging processes.

CONCLUSIONS

Changes in nomenclature, advances in diagnoses, gains in surgical technique operate to modify the validity of statistical statements. Recognizing the weakness of a statistical presentation there can be little question that the morbidity and mortality rates of this country are undergoing marked changes at all age levels. There is ample evidence that certain forms of disease have increased proportionately to population increase or decrease, and that a few diseases, particularly those related to the gastrointestinal system, show an excessive mortality ratio despite our gains in medical and surgical therapy. The gastrointestinal and the circulatory diseases, along with cancer and automobile accidents, constitute a present day challenge which cannot be ignored. The physician and surgeon alike are concerned with the prevention of unnecessary death which means all except those over age 60.

REFERENCES

1. Collins, S. D. Incidence and cause of illness at specific ages. *Milbank Mem. Quart.*, 13: 320–338, 1935.
2. Rolleston, Humphrey. Changes in character of disease. *Brit. M. J.*, March 25, 1933.
3. Metropolitan Life Insurance Company. Health in Boston a century ago. *Statist. Bull.*, 15: 2 (April) 1934.
4. City of New York Dept. of Health. *Quart. Bull.*, 2: 1, 1934.
5. Introduction to the Vital Statistics of the United States 1900–1930, p. 37. Washington, D. C., 1933.
6. New York State Dept. of Health. Division of Vital Statistics. Fifty-third Annual Report, vol. 2, 1932.
7. City of New York Dept. of Health. *Quart. Bull.*, 3: 64, 1935.

GASTROSCOPY AS AN AID IN THE DIAGNOSIS OF CARCINOMA OF THE PANCREAS

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CARCINOMA of the pancreas is one of the most difficult of the upper abdominal lesions to diagnose, especially if not associated with jaundice or a palpable tumor mass. From a diagnostic standpoint it is therefore fortunate, as Clute has pointed out, that most carcinomas of the pancreas occur in the head of the organ and, consequently, are associated with jaundice. Eusterman and Wilbur, however, have found from a review of 403 proved cases of carcinoma of the pancreas that one may anticipate an absence of jaundice in more than 20 per cent.

It is in this latter group of patients that the physician's diagnostic skill is sorely tried. Carcinoma of the pancreas should be suspected in any individual of middle age who complains of a deep-boring upper abdominal pain of short duration, relieved by bending forward, and associated with a rapid loss of weight. Confirmatory evidence, however, is very sparse. An epigastric mass is extremely significant but it is more often absent than present. Eusterman and Wilbur have aptly stated that, in spite of the stress that has been placed on the presence of glycosuria and steatorrhea in the diagnosis of carcinoma of the pancreas, these findings are conspicuous by their absence. Changes in the pancreatic ferments are infrequently present, and when present are of doubtful value. The same may be said of study of the gastric contents. The procedure that, up to the present, has probably been of the greatest value in the recognition of carcinoma of the pancreas is roentgenographic study of the gastrointestinal tract. While roentgenograms often suggest involvement of the pancreas, more often the diagnosis must await exploratory laparotomy.

It is, therefore, readily apparent that any procedure which might assist in earlier and more accurate recognition of such a highly fatal disease is worthy of report. It has been our good fortune to find gastroscopy of value in the diagnosis of two cases of carcinoma of the pancreas in the absence of jaundice or of a palpable tumor mass, and we feel that this is worthy of report, as it may be of value to other observers in arriving at an accurate and earlier diagnosis of this malignant disease.

That gastroscopy should, theoretically, be a suitable procedure as a diagnostic aid would seem apparent from a review of the anatomic relationship of the pancreas to the stomach. The anterior surface of the body of the pancreas forms a considerable portion of the bed of the stomach. Theoretically, any involvement of this portion of the pancreas should cause a change in contour of the adjacent stomach wall which should be recognized gastroscopically and lead to a correct diagnosis. (Fig. 1.) That this does occur is seen from the two following case reports.

CASE 1. A man 56 years of age first came under our observation at The Mayo Clinic in June, 1937 because of attacks of pain in various joints of his body. These attacks had been present in cycles during the preceding thirty years. A diagnosis of gout and obesity was made, the patient was placed on a purine-free diet and cinchophen was administered. He made definite improvement and was permitted to return home. He did very well on this regimen except for one severe attack of gout. In October, 1937, while away from home on a business trip and under considerable nervous strain, he took cinchophen one night without water. He awoke the following morning with a dull pain in the epigastrium, which was ascribed to the cinchophen. The pain con-

tinued, gradually becoming more severe and constant, so that he was unable to work or rest. Two weeks later the pain began to extend into the right renal region. There was no associated nausea, vomiting, or urinary symptoms. His appetite rapidly failed and there was a marked loss of weight. There were no tar-colored stools. The patient obtained relief by assuming a sitting position.

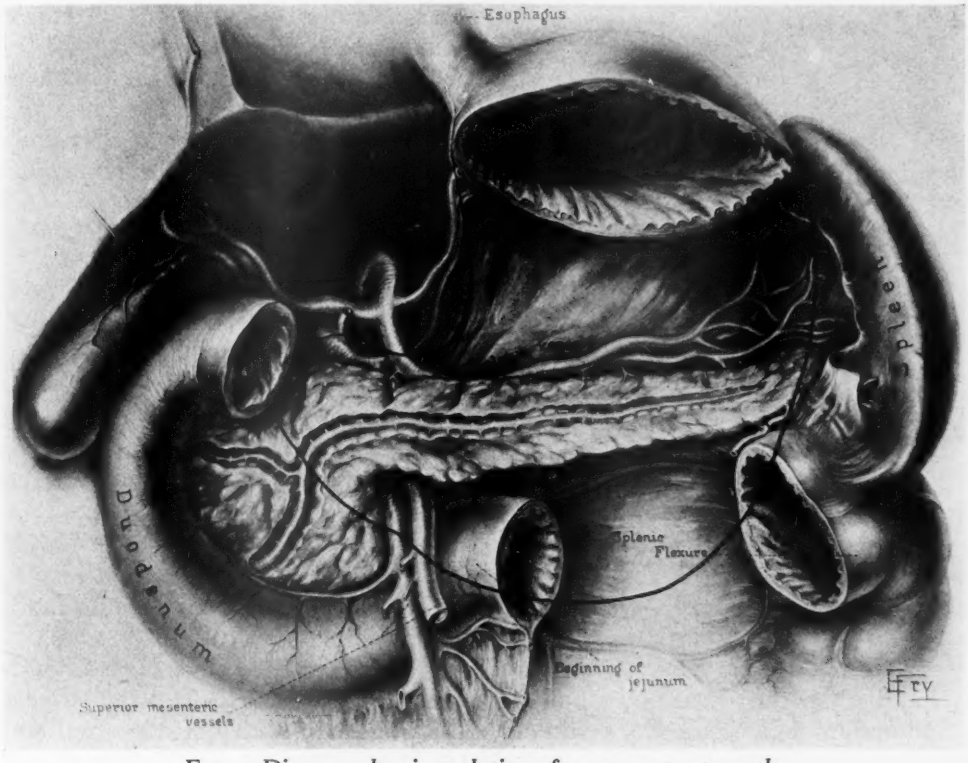

Fig. 1. Diagram showing relation of pancreas to stomach.

He returned to The Mayo Clinic on November 11, 1937 for further examination, just a month following the onset of his pain in the epigastrium. Physical examination revealed that he was well developed and well nourished but obviously he had lost considerable weight. The only physical finding of any significance was tenderness in the epigastric region; no tumor mass was palpable. Laboratory studies revealed the following results:

The concentration of hemoglobin was 14.1 Gm. per 100 c.c. of blood, and there were 4,530,000 erythrocytes and 5,800 leucocytes to the cubic millimeter of blood. The Wassermann reaction of the blood was negative, as were the results of the urine examination. The sedimentation rate was 32 mm. per hour. The concentration of blood sugar was 87 mg. per 100 c.c., and that of blood uric acid was 5.2 mg. per 100 c.c. Examination of the stools for occult blood and fat gave negative results. Studies of the liver function, gastric contents, amylase and lipase in the serum failed to reveal anything of importance. Roentgenographic studies of the kidneys, ureters, urinary bladder, stomach, and colon gave essentially negative results. A roentgenogram of the small bowel was not satisfactory. Roentgenographic examination revealed a poorly functioning gallbladder. This, however, did not seem adequate to account for the patient's symptoms. Intravenous urograms, as well as cystoscopic studies, were made to rule out the kidney as a possible source of difficulty, but these all gave negative results.

The patient's pain became progressively worse and it became necessary to resort to morphine for its control. A neurologic examination failed to reveal anything of diagnostic importance.

The severity of the epigastric pain, its recent onset in a man of 56 years of age, and the progressive loss of weight and strength, together with the absence of demonstrable lesions of the upper urinary and digestive tracts and of

the central nervous system, comprised a combination of findings pointing with a fair degree of certainty to the pancreas as the seat of a

Fig. 2. Gastroscopic appearance of bulging of mucosa produced by pancreatic tumor.

neoplastic growth. There was little, however, from the physical or laboratory side to confirm such a diagnosis.

With this information at hand, the patient was examined with a gastroscope on November 24, 1937 and the following conditions were observed (Fig. 2): On the posterior wall of the stomach, above the angle, there was a bulging forward of the mucosa into the lumen of the stomach which could not be obliterated by inflation of the stomach with air. There were also stiffening and thickening of the mucosa over the region involved. It was uncertain whether the tumor mass originated in the gastric wall itself or extragastrically, pushing the stomach wall forward. It seemed to us, however, that the condition was most likely a tumor originating in the pancreas.

With this information, a tentative diagnosis of carcinoma of the body of the pancreas was made and exploratory laparotomy advised. This was performed on December 2, 1937 by Dr. Pemberton, who found a mass larger than a fist involving the body and the tail of the pancreas. It was firm in consistency and nodular in character. An aspirating needle was inserted into the mass and a specimen obtained, which on microscopic examination was diagnosed as malignant. The gall-bladder was also examined and found to be large and flaccid, but no stones could be felt.

The patient continued to fail and died February 3, 1938. The findings at necropsy confirmed the diagnosis of carcinoma.

Case II. A woman 58 years of age first came to the clinic January 3, 1938, complaining of indigestion. She stated that she had enjoyed good health until three months before. At that time she had first noticed a dull, constant, aching pain in the lower left anterior portion of the chest, which was severe enough to keep her awake at night. Six weeks later the pain had become intermittent in character and had shifted to the midepigastric region. It was aggravated by eating and somewhat relieved by belching. With the shift in location of the pain there was also projection along both costal margins and then to the left shoulder blade. The patient was frequently awakened at night by the pain, and would have to get up and walk the floor. There was no nausea or vomiting, but there had been a loss of appetite and a decrease of 30 pounds (14 kg.) in weight during the month preceding our examination. At no time were there clay-colored or tar-colored stools, chills, fever or jaundice.

On physical examination the patient was found to be well developed, but she had obviously lost weight. She weighed 150 pounds (68 kg.), as compared with 180 pounds (82 kg.) one month previously. The blood pressure was 122 mm. of mercury systolic and 80 diastolic, the pulse rate was 74 per minute, and the temperature was 98.4°F. The results of physical examination were essentially negative. The results of urinalysis, the concentration of hemoglobin, erythrocyte and leucocyte counts, and the flocculation reaction for syphilis were all essentially normal. Gastric analysis failed to reveal anything abnormal. Roentgenographic studies of the gall-bladder, stomach and colon gave negative results. Electrocardiographic tracings also were normal.

Because of the location and character of the pain, gastroscopy was advised. This was performed January 6, 1938, under local anesthesia. At the junction of the middle and upper thirds of the stomach the posterior wall of the stomach was pushed forward and appeared nodular in character. The gastric mucosa overlying this region seemed infiltrated and it was impossible to say whether the condition was a beginning infiltration or gastritis.

Because of these findings and the character of the patient's symptoms, surgical exploration was advised; it was carried out on January 15, 1938 by Dr. Walters. On opening the abdomen, a carcinomatous nodule 5 cm. in diameter was

found in the lower margin of the left lobe of the liver. The primary lesion appeared to rise from the tail of the pancreas and was the size of a fist. The portion of the stomach in contact with the tumor was not involved in the malignant process but appeared to be involved in gastritis. The patient recovered from the exploration but died at home several months later.

It is readily apparent that gastroscopy is of value only in the recognition of tumors involving that portion of the pancreas that comes in contact with the stomach. This, however, is precisely the region in the pancreas in which it is most difficult to arrive at an adequate diagnosis. Whether gastroscopy may be of value in the diagnosis of carcinoma involving the head of the pancreas is very doubtful, and we have had no experience with such lesions gastroscopically. As a rule, however, lesions located in that portion of the pancreas give rise to jaundice, which attracts attention to the correct underlying disease.

While two cases are hardly sufficient to permit one to discuss differential diagnosis, there are two conditions seen gastroscopically that might simulate a tumor of the pancreas. The first is an exaggerated anterior curvature of the lumbar segment of the vertebral column, which may cause a bulging forward of the posterior wall into the lumen of the stomach. This, however, does not produce a nodular type of bulging and does not have an associated thickening of the overlying gastric mucosa.

The second condition that may simulate carcinoma of the body of the pancreas gastroscopically is a cascade type of stomach. Here likewise, owing to the sharp angulation of the stomach on itself, one gets the impression of something bulging forward into the lumen. On inflation of the stomach with air this bulging tends to disappear, which is not true of bulging due to an underlying tumor.

There is no reason, of course, why enlarged retroperitoneal glands might not give rise to a gastroscopic picture similar to that seen in tumor involving the body of the pancreas.

Whether further experience will demonstrate that gastroscopy is of greater value than other diagnostic procedures in cases of carcinoma of the body of the pancreas must await further observation and study.

REFERENCES

1. Clute, H. M. The problem of cancer of the pancreas. Chairman's address. *J. A. M. A.*, 107: 91–97 (July 11) 1936.
2. Eusterman, G. B., and Wilbur, D. L. Primary malignant neoplasm of the pancreas. A clinical study of eighty-eight verified cases without jaundice. *South. M. J.*, 26: 875–883 (Oct.) 1933.

PEDUNCULATED GASTRIC TUMORS

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PEDUNCULATED gastric tumors are responsible for some of the most interesting symptom complexes encountered in the upper abdomen. These lesions are not common, but can by no means be considered rare. Consideration in differential diagnosis that such a lesion may be present, together with the increasing skill of the radiologist in visualizing these tumors, has resulted in removing them from the group of pathologic curiosities.

INCIDENCE

Exact figures on the incidence of pedunculated gastric tumors are difficult to obtain. Many isolated reports of such lesions, presenting varying types of pathology are to be found in the literature, but few have had the privilege of studying any sizable group of cases. The incidence of gastric tumors which are benign has been estimated by several different writers: Minnes and Geschickter[1] 4.5 per cent; Eusterman and Senty[2] 1.3 per cent; Douglas[3] 2.9 per cent. Eusterman and Senty, in 1922, reported twenty-seven cases of benign gastric tumor observed over a fourteen-year period at The Mayo Clinic, of which 25 per cent were capable of blocking the pyloric outlet. During the same period 2,168 patients with gastric carcinoma were operated upon, and an additional group of 2,285 cases of gastric malignancy were denied surgery due to the extent of the disease. Balfour and Henderson[4] have observed fifty-eight cases of benign gastric neoplasm, of which 10 per cent were sufficiently mobile to produce pyloric obstruction. Eliason, Pendergrass and Wright,[5] in 1926, collected 611 cases of benign gastric tumor, of which forty-eight, or 7.5 per cent, were pedunculated. These authors personally studied a series of fifty benign gastric tumors of which ten, or 20 per cent, were pedunculated.

ETIOLOGY

The cause of solitary pedunculated gastric tumors remains as obscure as that of neoplasms in general. The vast majority, however, must take their origin as simple, benign, epithelial tumors in the gastric mucosa, or as mesothelial tumors in the submucosal tissues. With growth and extension into the lumen of the stomach the expulsive action of the gastric peristalsis attempts to extrude them toward the pylorus and results in the development of a definite pedicle. Eliason and Wright[6] suggest that these new growths take their origin in a low grade inflammatory reaction in the mucosa resulting from chronic irritation. Johnson[7] believes that chronic alcoholism, chronic gastritis, atheroma of the gastric vessels, and conditions leading to chronic nutritional disturbances of the stomach are responsible. This author feels that chronic gastritis is probably the most common etiologic factor, resulting in hypertrophic thickening and lobulated swelling of the gastric mucosa, which leads to the development of the polypoid formation. The common association of an achlorhydria, together with the relatively high incidence of gastric polyps in proved cases of primary pernicious anemia coming to post-mortem, would tend to support this suggestion.

In the instance of diffuse gastric polyposis or adenopapillomatosis gastrica, it is possible that other factors play a part. Brunn and Pearl[8] suggest that this condition may arise either on a congenital or a

familial basis, due to fetal epithelial anlage plus chronic inflammation, or simply as a result of prolonged chronic inflammation of the gastric mucosa.

PATHOLOGY

From a gross pathologic standpoint pedunculated lesions of the stomach may be classified under four major headings: (1) solitary benign gastric tumors which, as a result of growth and gastric motility, develop pedicles; (2) malignant pedunculated gastric tumors, usually representing carcinomatous degeneration of a preëxisting benign polyp or adenoma; (3) diffuse gastric polyposis, adenopapillomatosis gastrica, or gastritis polyposa; and (4) prolapsing prepyloric gastric mucosa.

Solitary benign gastric new growths may appear as myomata, papillomata, polypi, myxomata, adenomata, cysts, lipomata or fibromata. Minnes and Geschickter have divided all benign gastric tumors into four groups according to the tissue from which they take their origin: epithelial, mesenchymal, endothelial, and a mixed group including cysts. In a series of 931 collected cases they found that the epithelial tumors comprised 35.2 per cent, the mesenchymal tumors 65.9 per cent, and the other two groups 10 per cent. Eliason and Wright[6] found that in their series of fifty cases, papillomata and polypi represented 64 per cent of the group, with adenomata next in frequency (10 per cent). Balfour and Henderson report that 24 per cent of their group of fifty-eight benign gastric tumors were fibro-adenomatous polypi, and 40 per cent were fibromata, myomata and myofibromata. In Eusterman and Senty's series of twenty-seven benign gastric tumors, myomata were the most common (38 per cent), with fibromata and polypi next in order (30 per cent).

The striking frequency with which these solitary benign gastric lesions are found in cases diagnosed as typical primary pernicious anemia is worthy of note. Brown,[9] in a series of 151 cases of primary pernicious anemia studied at post-mortem, found gross lesions in the gastrointestinal tract in eighty-two. There were thirteen benign tumors in this series, twelve in the stomach and one in the jejunum. Four of these were myomas, eight were epithelial polyps and one was a mixed tumor.

These benign tumors of the stomach may vary in size from a few millimeters up to rather massive dimensions. Bland-Sutton[10] reports a myoma weighing 7 pounds.

Gastric fibromata, adenomata and simple polypi tend more commonly to arise in the body and pyloric end of the stomach. They are usually found to take their attachment from the anterior or posterior surfaces of the stomach and more rarely from the curvatures. With continued growth they project into the gastric lumen and tend to develop their pedicles as a result of peristaltic activity. The average length of the pedicles in the reported cases has been 2 cm., but many of these become much longer as the tumor increases in size. Their proximity to the gastric outlet results in the frequent production of pyloric obstruction, usually of a ball-valve, intermittent type. Balfour and Henderson observe that this occurs in approximately 10 per cent of the solitary benign gastric neoplasms. The other frequent complications resultant from these solitary benign tumors are intussusception, ulceration, gastric hemorrhage and malignant degeneration.

Malignant pedunculated gastric tumors usually represent examples of carcinomatous degeneration in a preëxisting adenoma, polyp or papilloma. The incidence of malignant degeneration in these lesions has been varyingly estimated by different authors: Stewart[11] 28 per cent; Brunn and Pearl[8] 12 per cent; Myer and Brams[12] 60 per cent; Miller, Eliason and Wright[13] 35 per cent, and Benedict and Allen[14] 41.2 per cent. McRoberts[15] found definite malignant degeneration in the epithelium about the periphery of the pedicle in four out of five gastric polyps. Pennington[16] has also pointed out that it is in the gastric mucosa adjacent to the pedicle that neo-

plastic change is usually first evident. Walters[17] calls attention to the interesting clinical fact that in diffuse gastric polyposis malignancy is not the rule, while in solitary gastric polypi malignancy occurs with great frequency.

The presence of a definite gastric carcinoma separated by normal mucosa from an associated benign polyp has been reported by Douglas.[3] This patient succumbed after a recurrence fifteen months after a gastric resection. The case which we are reporting presented very similar findings at operation. Cases of this type demonstrate the narrow margin that exists between an apparently benign gastric polyp or adenoma and a gastric malignancy. This is responsible for the surgical dictum that all gastric polypi should have the advantage of radical surgery if feasible. Sinclair,[18] after a study of these cases, concluded that "adenomatous polypi of the stomach, whether single or multiple, must be accepted as definitely precancerous lesions, but the available evidence goes to show that the association is much less intimate than in the case of the large intestine."

Diffuse gastric polyposis or adenopapillomatosis gastrica represents a rare type of gastric lesion, characterized by the presence of multiple pedunculated or polyp-like tumors. Higgins[19] divides this group of tumors into three distinct types from a pathologic standpoint: (1) polyadenoma polypeux—consisting of polyps with multiple cysts, possibly due to obstructed ducts; (2) polyadenoma en nappe—with numerous polyps in clumps or plaques; and (3) polyadenoma of Brunnerian type—containing duodenal glands (quite rare).

Mills[20] was able to collect fourteen cases of multiple gastric polypi from the literature prior to 1922. In this series the number of polypi varied from six to over 250. He could find no evidence of inflammation in the mucosa between the tumors and does not believe that an inflammatory reaction is important in their production. Strauss, Meyer and Bloom,[21] in 1928, reported two cases of polyadenoma en nappe, bringing the reported cases of this type to seven. In both of their personal cases the lesion was primarily limited to the fundus of the stomach and characterized clinically by gastric discomfort, repeated gastric hemorrhages, and abnormal amounts of gastric mucus of an egg-white consistency.

The two major complications that may develop with this type of gastric polyposis are hemorrhage and malignant degeneration, although the latter is not nearly so common as in the instance of a solitary prepyloric gastric polyp. Oshsner and Moser[22] have recently reported two cases of multiple gastric polypi, one succumbing with metastasis and the other with massive gastric hemorrhage. Joyce and Diack[23] have also observed a patient with diffuse gastric polyposis treated by a Polya type of gastric resection; this individual succumbed eighteen months later from metastasis.

Prolapsing gastric mucosa must be considered for the sake of completeness in any discussion of pedunculated gastric tumors. This condition is not in any way neoplastic, but represents a redundancy of the prepyloric mucosa of a varying degree, prolapsing through the pyloric ring with the development of intermittent pyloric obstruction. Pendergrass and Andrews[24] report eight cases of this condition diagnosed by *x*-ray, with subsequent confirmation on surgical exploration in seven instances. The condition clinically and roentgenologically may simulate the picture presented by a pedunculated gastric polyp and these authors state that in their experience it is often impossible to differentiate between the two by *x*-ray examination.

SYMPTOMS

The majority of patients with pedunculated gastric tumors are males between the ages of 40 and 65. Monat,[25] however, observed a 12 year old girl with a gastric intussusception incident to a gastric polyp, and isolated cases have been reported in almost every decade of life.

Pedunculated tumors in the body of the stomach produce symptoms varying with their location. Those near the pylorus may produce intermittent ball-valve obstruction with attacks of severe epigastric pain, nausea and vomiting. This obstruction is usually transient and is relieved when the tumor falls back into the body of the stomach. Instances are recorded in which the patient was conscious of something slipping or moving in the epigastrium just before the beginning of an attack, as noted in our patient. These patients often find that changing their position, or taking fluid will dislodge the obstructing tumor mass and give relief.

Those tumors situated sufficiently far from the pylorus to prevent the development of intermittent obstruction may only produce symptoms of vague epigastric discomfort of an indefinite type. Gossett et al.[26] found that in twenty-seven of a series of sixty-six collected cases of pedunculated gastric tumors there were no symptoms to attract attention to the stomach. These patients usually seek medical relief because of a sudden gastric hemorrhage which may be massive enough to be fatal, or because of weakness resulting from chronic blood loss. The bleeding in these cases results from either torsion of the pedicle with partial strangulation, or more commonly from an ulcer which develops on the surface of the pedunculated tumor.

It is interesting to note that some of these patients who bleed slowly over a period of time develop glossitis, achlorhydria, diarrhea and a blood picture which is indistinguishable from primary pernicious anemia. Waters[17] reports three cases of bleeding gastric polyps occurring in patients with primary pernicious anemia, all successfully removed with improvement. Priestley and Heck[27] observed three cases of typical primary pernicious anemia, with established neurologic signs, in each of which on routine gastrointestinal examination there was found a bleeding gastric polyp. All three were definitely improved by surgical removal of the polyp.

Isolated cases have been reported by different authors in which the only symptoms and physical findings elicited were those incident to profound anemia, with or without vague epigastric symptoms, and on routine gastrointestinal studies a gastric polyp was discovered. These observations emphasize the importance of a thorough *x*-ray study of the upper gastrointestinal tract in every case of severe anemia, including those which appear to be clear-cut cases of primary pernicious anemia.

In addition to obstruction, ulceration, and hemorrhage, the other common complication encountered with pedunculated gastric tumors is intussusception. Iceton et al.[28] report a neuroblastoma of the gastric wall producing a fatal intussusception through the pylorus in a 47 year old female. Other instances of gastroduodenal and gastrojejunal intussusception have been reported by Wade,[29] Monat,[25] Barnett,[30] and Shuman and Cruikshank.[31]

DIAGNOSIS

The symptomatology of pedunculated gastric tumors is so varied that no one picture can be considered diagnostic. However, these lesions should be kept in mind and suspected when a history is elicited of vague epigastric distress with bouts of intermittent pyloric obstruction, especially if accompanied by achlorhydria and anemia. A routine physical examination is rarely of assistance in establishing the diagnosis, for the vast majority of these tumors cannot be palpated. A careful *x*-ray examination by a competent radiologist is the chief method of diagnosis in each instance. Lockwood[32] states that the principal sign of a mural, sessile or pedunculated growth in the stomach is the round or oval shaped filling defect seen radiographically. Peristalsis is not interrupted and the tumors can usually be shifted about in the stomach. Kirklin and Broders[33] caution that the stomach should

be absolutely empty before the examination is undertaken and that a small amount of thin barium be used to permit accurate visualization of the gastric contours. They found that food masses, simple hypertrophy of the gastric mucosa and nonulcerated polypoid carcinoma were very easily confused with pedunculated gastric new growths. Pendergrass[34] and Rigler[35,36] have outlined the most common *x*-ray findings seen in the examination of these patients. These have been summarized by Pendergrass[34] as follows:

1. A filling defect not easily demonstrated by fluoroscope. When seen it is sometimes possible to palpate the lesion.

2. Peristalsis is not often interrupted unless there is an associated carcinoma with infiltration of the gastric wall.

3. A six-hour gastric residue is usually present.

4. A central filling defect in the duodenum is seen on *x*-ray in the prone and right oblique positions.

5. No defect is noted in the stomach in the pyloric region. This is important in differentiating prolapsing tumors from prolapsing prepyloric mucosa.

La Roque and Shiflett[37] observe that in general it may be assumed that "a filling defect suggesting a new growth in the duodenal bulb, accompanied by a six-hour residue, is indicative of a prolapsing tumor, while the absence of the six-hour residue is indicative of the tumor arising in the duodenum itself."

The most common lesions which may be confused in the differential diagnosis include gastric and duodenal ulcer, gastric carcinoma, prolapsing gastric mucosa, mesenteric bands and hypertrophied pyloric muscle in the adult. A careful evaluation of the history and physical findings may give some assistance in differentiation, but the principal burden falls upon the radiologist. Those who have had experience with this type of lesion are capable of correctly diagnosing their presence in a high percentage of cases and it is upon the radiologist that the surgeon must depend.

TREATMENT

The treatment of prolapsing gastric tumors is surgical. The tendency of these new growths to ulcerate, bleed, obstruct and undergo malignant degeneration is well established. Surgical treatment should include a radical removal of the tumor together with the adjacent stomach, since the carcinomatous degeneration so frequently first develops about the base of the pedicle. It is quite true that in dealing with a simple pedunculated fibrinous polyp, local removal of the polyp might be adequate, but this type of procedure is in general a dangerous one to follow. Numerous reports are to be found in the literature of recurrence and subsequent death from metastasis following conservative surgical procedures in the treatment of these tumors. The wiser course is to follow the advice of Balfour[38] and subject these cases to partial gastrectomy rather than a local excision if the patient's condition permits, and in this way remove the local area of the stomach and assure safety from recurrence.

Since the majority of these solitary prolapsing gastric polyps arise in the body or prepyloric region of the stomach, some form of partial gastrectomy will completely remove the entire area of stomach under suspicion. When one encounters a bleeding pedunculated tumor in the cardia of the stomach however, local excision with removal of as much of the adjacent mucosa and gastric wall as is feasible is the only course possible.

It is worthy of note that often the operating surgeon will be unable to demonstrate the presence of the pedunculated tumor through the gastric wall when the abdomen is first opened. They have a tendency to be very elusive, depending upon the size and the length of their pedicles. Accordingly they may be found anywhere from the cardia to the second or third portions of the duodenum. If the diagnosis of a pedunculated tumor has been made by a competent radiologist, its absence cannot be definitely established until the stomach

has been opened and the entire inner surface carefully examined under direct vision. Only if this step has been carried out is one justified in assuming that the tumor is not there and an error has been made in diagnosis.

The following case, recently observed, demonstrates most of the prominent features that characterize these lesions. It is of interest in that a correct diagnosis was made by the radiologist of a prolapsing prepyloric gastric tumor before operation and the presence of an associated gastric carcinoma in the body of the stomach was also suspected.

CASE REPORT

J. W., a white male farmer, 58, was seen in consultation with Dr. Frank Conlin at St. Catherine's Hospital on February 4, 1937. The patient was admitted with an initial complaint of recurrent attacks of epigastric pain for a period of six months.

There was no history of any previous serious illnesses. The patient had done hard manual work until the onset of the present illness and always considered himself to be in excellent health.

Six months before admission the patient first became aware of what he described as a "weak" feeling in his epigastrium, unassociated with any pain. He then noted the development of rather severe attacks of epigastric pain, beginning in the mid-epigastrium and radiating to each side of the midline for a distance of 15 cm. This pain was dull in type, usually came on at 3 or 4 o'clock in the morning, and was promptly relieved by sitting up in bed or drinking a glass of milk. After eating his morning meal the patient was able to do his day's work without any discomfort. Pain was rarely experienced during the day, but occasionally the patient noted a sensation of "weakness" in the pit of the stomach. Coarse foods were especially prone to produce this sensation but no other foods were troublesome. Occasionally something seemed to move or slip in the epigastrium and following this sensation pain would develop about the pylorus. Three months before admission the patient was operated upon elsewhere for a rectal fistula. He had some bright red blood in his stools prior to this operation but none since. There had been no nausea or associated vomiting, and no change had been noted in the bowel habit. The patient denied any loss of weight or strength. The

Fig. 1. *X*-ray examination of stomach demonstrating pedunculated gastric polyp in prepyloric region and defect on greater curvature resultant from coëxistent gastric carcinoma.

major complaint was the periodic attacks of epigastric pain which gradually increased in severity and frequency during the previous four months.

The patient was well developed and in a good state of nutrition. He did not appear to have lost any weight. The teeth were in poor condition, the tonsils small and the throat slightly injected. There were no palpable glands in the neck. The chest was clear on percussion and auscultation. The heart was not enlarged, the sounds regular and of good quality, with no murmurs. Blood pressure was 140/90. There was no abdominal distention and no scars or hernia were present. There was slight but definite resistance over the epigastrium on palpation and a little deep tenderness was demonstrable over this region. No masses could be made out. The extremities and reflexes were all normal.

Laboratory Data.

Blood hemoglobin 90 per cent R. B. C. 5,000,000. W. B. C. 6,100.

Differential: Polys 55 per cent (staph. 1 per cent, seg. 54 per cent); lymphocytes 38 per cent, eosinophiles 7 per cent.

Urine: Specific gravity 1.007; no albumin, sugar or acetone; occasional white blood cells.

Wassermann negative.

Stool examination: Negative for occult blood and for parasites on three occasions.

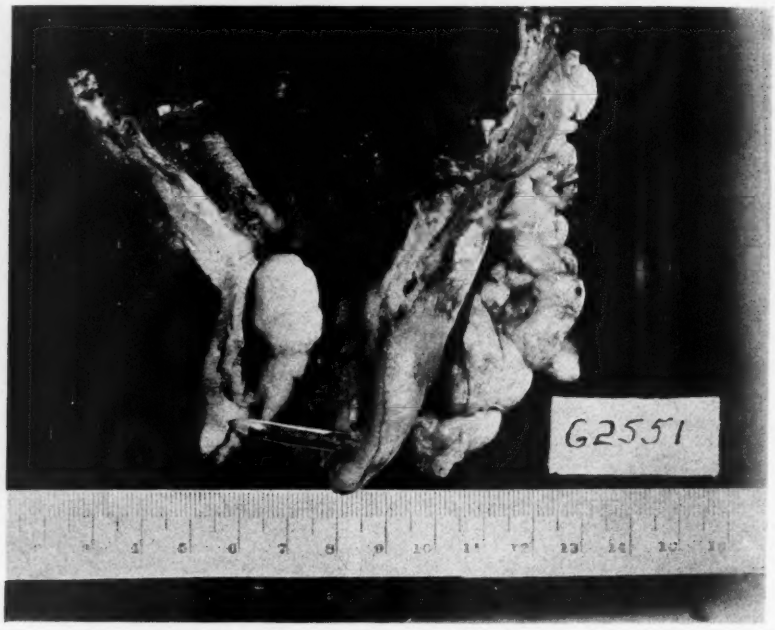

FIG. 2. Resected portion of stomach opened to show the size and shape of the gastric polyp and attachment of its pedicle.

Gastric Analysis:

	Minutes			
	15	30	45	60
Free	0	0	0	0
Total	32	14	10	10

A gastrointestinal examination showed that the stomach filled poorly at the pyloric end and disclosed a constant defect visible there and at the base of the duodenum, which looked like that produced by a pedunculated polyp arising in the adjacent pylorus. (Fig. 1.) At five hours the stomach was practically empty. The small bowel showed no abnormality. At twenty-four hours the colon was irregularly filled with the meal. A barium enema showed no apparent lesion in the colon.

A recheck of the stomach after twenty-four hours showed essentially the same picture seen at the first examination. The roentgenologist concluded that there was a pedunculated tumor in the duodenum, probably arising in the prepyloric stomach. The adjacent pylorus appeared irregular, suggesting the possible presence of a carcinoma in this area in addition to the polyp.

Operation. Under avertin and open drop ether, the abdomen was opened through a right rectus incision. Examination of the stomach showed a typical area of carcinoma on the greater curvature and anterior surface, 4 cm. in diameter. This lesion was 3 inches proximal to the pylorus and had invaded the peritoneal surface of the stomach. Palpable within the prepyloric stomach was a pedunculated intragastric tumor, which could be pushed through the pylorus into the duodenum for a distance of 3 cm. and back into the body of the stomach. This tumor seemed to be about 2 cm. in diameter and when prolapsed into the duodenum effectively blocked its lumen. There was no evidence of metastasis in the liver, the regional gastric nodes or pelvic peritoneum.

The gastric polyp was replaced into the prepyloric stomach and a gastric resection carried out, removing the distal half of the stomach well above the carcinoma, together with the pyloric ring. A Billroth type I reconstruction was then carried out, employing the modification suggested by Horsley. The anastomosis was completed without tension and the patient returned to his room in excellent condition.

Convalescence was entirely uneventful. A postoperative ulcer diet was given five days after operation. The patient was dismissed from the hospital on the fifteenth postoperative day. He remained entirely well for fourteen months, and is doing hard work on his farm with no complaints.

Pathologic Report. The specimen consisted of the distal half, together with the pylorus of the stomach. (Fig. 2.) The mucosa was somewhat atrophic and showed slight injection. On the postero-inferior surface of the stomach 3 cm. proximal to the pylorus was a polyp 2.5 cm. in diameter, attached to the gastric mucosa by a stalk-like pedicle 3 cm. long. The mucosa over the polyp was smooth with no visible necrosis or ulceration. Situated well above the polyp, and entirely independent from it, was an area of carcinoma 4 cm. in diameter, lying on the greater curvature and anterior surface of the stomach. It was firm and plaque-like, and its mucosal surface showed no gross ulceration. The peritoneal covering of the stomach had been invaded but there was no evidence of regional metastasis. No other polypi were visible in the resected specimen.

The microscopic diagnosis was scirrhous carcinoma of the stomach, fibrinous pedunculated gastric polyp, the mucosa about the base of the polyp showed no evidence of malignant change.

CONCLUSIONS

1. Pedunculated gastric tumors cannot be considered rare lesions.

2. From 10 to 20 per cent of benign gastric neoplasms develop pedicles and are capable of producing pyloric obstruction.

3. These pedunculated tumors show evidence of malignant degeneration in from 30 to 40 per cent of the cases.

4. Vague epigastric distress associated with intermittent attacks of pyloric obstruction represent the most common clinical features.

5. Ulceration, hemorrhage, obstruction and intussusception are the principal complications resultant from these lesions.

6. All cases of severe anemia of undetermined origin should be carefully studied to exclude the presence of a bleeding gastric polyp.

7. Proved cases of primary pernicious anemia should have the benefit of a thorough gastrointestinal study because of the frequent occurrence of polyps in this condition.

8. The final diagnosis rests in the hands of a competent radiologist.

9. Treatment should consist of partial gastrectomy, if this be feasible, to remove both the polyp and a wide area of the adjacent gastric wall.

REFERENCES

1. Minnes, J. F., and Geschickter, C. F. Benign tumors of the stomach. *Am. J. Cancer*, 28: 136, 1936.
2. Eusterman, G. B., and Senty, E. G. Benign tumors of the stomach. *Surg., Gynec. & Obst.*, 34: 5, 1922.
3. Douglas, J. Benign tumors of the stomach. *Ann. Surg.*, 77: 580, 1923.
4. Balfour, D. C., and Henderson, E. Benign tumors of the stomach. *Ann. Surg.*, 85: 354, 1927.
5. Eliason, E. L., Pendergrass, E. P., and Wright, V. W. M. X-ray diagnosis of pedunculated growths and gastric mucosa prolapsing through the pylorus. *Am. J. Roentgenol.*, 15: 295, 1926.
6. Eliason, E. L., and Wright, V. W. M. Benign tumors of the stomach. *Surg., Gynec. & Obst.*, 41: 461, 1925.
7. Johnson, C. A. Benign neoplasms of the stomach. *Am. J. Surg.*, 18: 324, 1932.
8. Brunn, H., and Pearl, F. Diffuse gastric polyposis. *Surg., Gynec., & Obst.*, 43: 559, 1926.
9. Brown, M. R. The pathology of the gastro-intestinal tract in primary pernicious anemia and subacute combined degeneration of the spinal cord. *New England J. Med.*, 210: 473, 1934.
10. Bland, S. J. Fibroids, lipomas, dermoids and polypi of the stomach and intestine. *Lancet*, 2: 5, 1920.
11. Stewart. Quoted by Benedict and Allen.[14]
12. Meyer, K. A., and Brams, W. A. Non-cancerous tumors of the stomach. *Surg., Gynec. & Obst.*, 41: 311, 1925.
13. Miller, T. G., Eliason, E. L., and Wright, V. W. M. Carcinomatous degeneration of polyps of the stomach. *Arch. Int. Med.*, 46: 841, 1930.
14. Benedict, E. B., and Allen, A. W. Adenomatous polypi of the stomach with special reference to malignant degeneration. *Surg. Gynec. & Obst.*, 58: 79, 1934.
15. McRoberts, J. W. Gastric polyps. *Proc. Staff. Meet., Mayo Clin.*, 8: 685, 1933.
16. Pennington, W. E. Gastric polyps. *J. Indiana M. A.*, 28: 226, 1935.
17. Walters, W. Gastric polypi. *Proc. Staff Meet., Mayo Clin.*, 8: 315, 1933.
18. Sinclair, N. Diffuse gastric polyposis. *Brit. J. Surg.*, 20: 645, 1933.
19. Higgins, C. Benign tumors of the stomach. *Ann. Surg.*, 81: 949, 1925.

20. Mills, P. Multiple polypi of the stomach. *Brit. J. Surg.*, 10: 226, 1922.
21. Strauss, A. A., Meyer, J., and Bloom, A. Gastric polyposis. *Am. J. M. Sc.*, 176: 681, 1928.
22. Ochsner, H. C., and Moser, R. H. Diffuse gastric polyposis. *J. Indiana M. A.*, 26: 427, 1933.
23. Joyce, T. M., and Diack, S. Gastric polyposis. *S. Clin. North America*, 13: 1417, 1933.
24. Pendergrass, E. P., and Andrews, J. R. Prolapsing lesions of the gastric mucosa. *Am. J. Roentgenol.*, 34: 337, 1935.
25. Monat, T. B. Two cases of polypus of the stomach. *Brit. J. Surg.*, 13: 165, 1925.
26. Gosset, A., Bertrand, I., and Loewy, G. Pedunculated tumors of the stomach. *J. de chir.*, 23: 577, 1924. Abst., *J. A. M. A.*, 83: 796, 1924.
27. Priestley, J. T., and Heck, F. J. Bleeding malignant polypoid lesions in cardia with severe anemia. *Am. J. Surg.*, 101: 839, 1935.
28. Iceton, S. G., Poate, H. R. G., and Tebbutt, A. H. T. Gastric intussusception in adult due to rare tumor. *M. J. Australia*, 1: 82, 1931.
29. Wade, H. Intussusception of the stomach and duodenum due to a gastric polypus. *Surg., Gynec. & Obst.*, 17: 184, 1913.
30. Barnett, L. E. A case of myoma of stomach with gastro-duodenal intussusception. *Brit. J. Surg.*, 12: 615, 1925.
31. Shuman, J. W., and Cruikshank, D. Duodenal intestinal obstruction due to gastric polyp. *New York J. Med.*, 117: 694, 1923.
32. Lockwood, B. C. Benign tumors of the stomach. *J. A. M. A.*, 98: 969, 1932.
33. Kirklin, B. R., and Braders, A. C. Roentgenologic and pathologic aspects of polyposis. *J. A. M. A.*, 98: 95, 1932.
34. Pendergrass, E. P. Prolapse of pedunculated tumors and gastric mucosa through the pylorus: X-ray diagnosis. *J. A. M. A.*, 94: 317, 1930.
35. Rigler, L. G. X-ray observation on benign gastric tumors. *Am. J. Surg.*, 8: 144, 1930.
36. Rigler, L. G. Roentgen observations on prolapsing benign gastric lesions. *Am. J. Roentgenol.*, 20: 529, 1938.
37. La Roque, G. P., and Shiflett, E. L. Benign tumors of the stomach. *Ann. Surg.*, 96: 240, 1932.
38. Balfour, D. Gastric polyps. *Proc. Staff Meet., Mayo Clin.*, 8: 685, 1933.

THIRTY-NINE CASES OF APPENDICITIS IN A SINGLE FAMILY PEDIGREE

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AUTHORS have attributed infections of the vermiform appendix to an imposing array of diverse causes. Outstanding among these alleged causes may be named anatomic defects of position, of form, of size, of vascularization, of attachment and presence of Gerlach's semilunar valve. Other suspect conditions are arthritis with a tendency to produce both gallstones and appendicular fecaliths; a constitutional diathesis conducive to autoinfection; a tendency for infection to settle in the lymphoid tissues including the appendix, following such diseases as scarlet fever or scarlatina, grippe and typhoid fever. Nervous constitutions producing constipation as well as other digestive disorders are accused. Intestinal parasites are also incriminated.

It seems very probable that most of the alleged causes are responsible for many cases, and hence it appears unwise to search for a single etiologic factor common to all appendicitis infections.

Except in the event of infection resulting from the presence of intestinal parasites, the immediate physical causes of appendicular infection mentioned above might be determined by or contributed to constitutional diatheses of one sort or another and, indeed, by consulting the literature one may find evidence that such diverse predisposing hereditary backgrounds do exist.

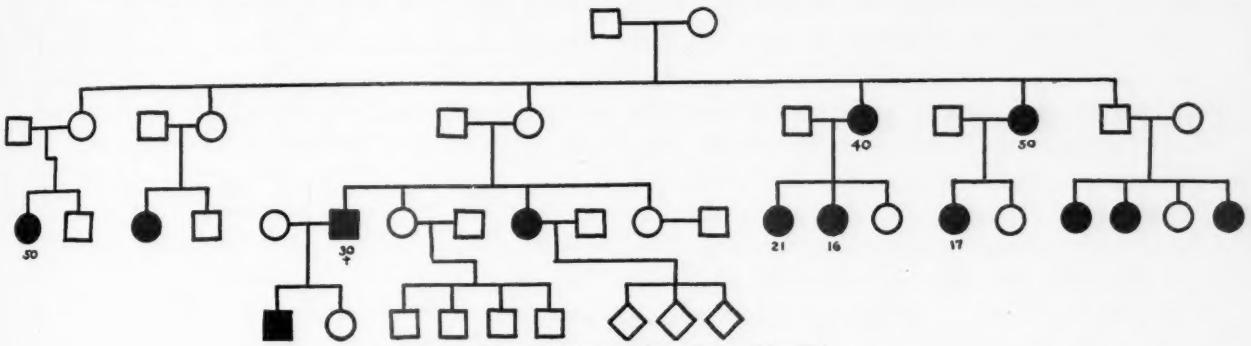

FIG. 1. Appendicitis. (Data from Caroli.)

Caroli (quoted by Carnot[1]) described the family charted in Figure 1* in which thirteen persons out of a total of thirty-six in four generations had appendicitis.

Okinczyc, quoted by Weinsaft,[2] describes the pedigrees of appendicitis shown in Figures 2, 3, and 4. The appendices of affected members of the family charted in Figure 3 were all between 15 and 17 cm. in length and were coiled.

Weinsaft reports the family charted in Figure 5, in which a daughter, a father and a paternal uncle were affected.

Colley[3] reports three small pedigrees whose affected members bore anatomic defects assumed to be the causes of infec-

[1] CARNOT, P. Les maladies digestives familiales. *Paris méd.*, 87: 288–299, 1933.

* In our charts (except Fig. 1) squares represent males, circles represent females. Numerals placed beneath the symbols indicate age at time of attacks.

[2] WEINSAFT, P. Les appendicites familiales (remarques etiologiques). Univ. Paris Thesis 1933.

[3] COLLEY, F. Beiträge zur Klärung der Frage von der Erblichkeit der Enzündung der Blinddarmanhanger. *Arch. f. Klin. Chir.*, 103: 177–208, 1914.

tion. In Figure 6 is represented a family the affected members of which lacked 1½ cm. of mesoappendix. However, contrary to the statement of Weinsaft, this does not represent a Mendelian characteristic, just because there are 3 affected to 1 normal in the third generation. Indeed, this anatomic peculiarity appears to be due to a single dominant gene, in which case the expectation is equal numbers of affected and normals. The 3 to 1 ratio obtained in this isolated instance was due to the probable error of random sampling. In Figure 7 are charted a father and a daughter in which the artery of the mesenteriole was very small and in which the vein was almost obliterated. In Figure 8 there was found at operation, an appendix fused to an ovarian cyst in a mother and also in her daughter.

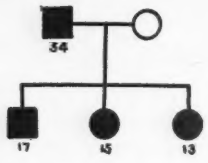

Fig. 2.

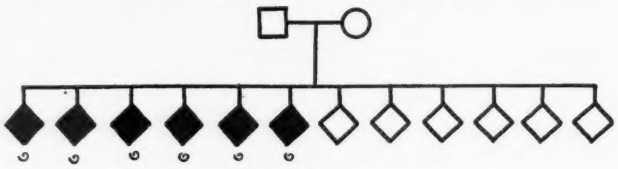

Fig. 3.

Fig. 4.

Figs. 2, 3, and 4. Appendicitis. The small coils in Figure 3 represent coiled appendices. (Data from Okinczyc.)

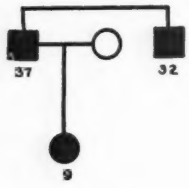

Fig. 5. Appendicitis. (Data from Weinsaft.)

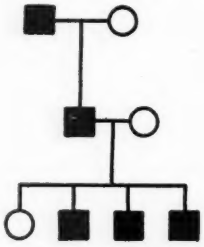

Fig. 6. Missing meso. (Data from Colley.)

Alvarez[4] supports the lithiasis theory with a pedigree shown in Figure 9.

Leckie[5] reported the pedigree of appendicitis which we have charted in Figure 10.

Short, thick appendices having wide openings into the cecum and subject to clogging with feces have been described by Balogh.[6] We show his material in Figure 11.

Van Meter[7] suggests inherited susceptibility to appendicular infections dependent upon inherited anatomic peculiarities. In Figure 12 we show Van Meter's pedigree of large angulated appendices. In Figure 13 we show Van Meter's data for coiled appendices.

Fig. 7.

Fig. 8.

Fig. 7. Small veins. (Data from Colley.)

Fig. 8. Appendix adhered to ovarian cyst. (Data from Colley.)

Magruder[8] describes four families in which more than one member had appendicitis. The largest pedigree (Fig. 14) shows twelve affected persons. Another family shown in Figure 15 has five affected members, while the other two pedigrees (Figs. 16 and 17) have merely two affected members.

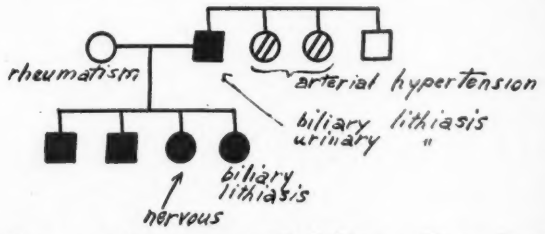

Fig. 9. Nervousness and lithiasis. (Data from Alvarez.)

[4] Alvarez, Hypertension and appendicitis in eight members of one family. *M. Clin. North America*, 2: 1435–1447, May 1928.

[5] Leckie, G. G. Interesting family history of appendicitis. *Canad. M. A. J.*, 36: 287, 1937.

[6] Balogh, E. Beitrag zur Lehre von der Erkrankung an appendicitis. *Deutsche Ztschr. f. Chir.*, 245: 325–328, 1935.

[7] Van Meter, S. D. Hereditary appendicitis. *Colorado Med.*, 17: 241–244, 1920.

[8] Magruder, E. M. Hereditary predisposition to appendicitis. *Virginia M. Monthly*, 45: 309, 1919.

In 1922 Cignozzi[9] summarized his findings in 500 cases of appendicitis within a circumscribed geographic area. He felt that when the appendix was situated high, due to incomplete rotation of the colon, attacks of appendicitis were more acute. Among these 500 cases fifty were found to be familial.

The authors have found several families affected by appendicitis. In Figure 18 is charted a family having five affected members, all operated on. Figure 19 describes a family having ten members that were operated for recurrent appendicitis. The "X" marks indicate burst appendices at the time of operation. The affected persons in this family all had retrocecal appendices.

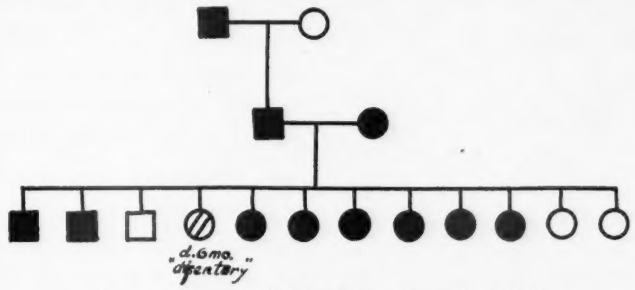

Fig. 10. Appendicitis. (Data from Leckie.)

dominant unit-characters. Such characters are: (1) missing section of mesoappendix; and (2) variations which may be described as coiled, short, long and retrocecal. It has been assumed that these bizarre morphologic forms produce appendicitis, but no one has operated on normal members of the same families to prove whether or not these individuals may bear these same anatomic abnormalities throughout life with impunity.

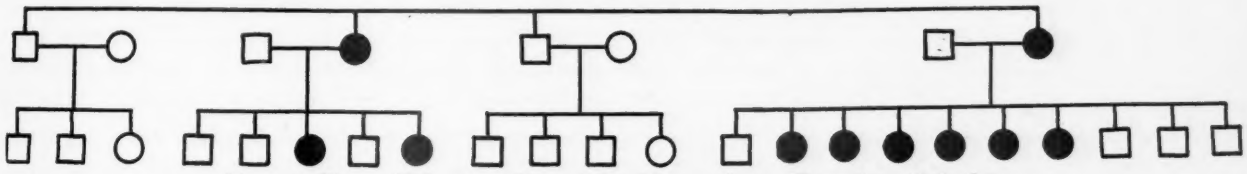

Fig. 11. Short thick appendices with wide openings. (Data from Balogh.)

That other disturbances of the digestive tract such as tonsillitis, gall-bladder disease, and ulcerative colitis are occasionally present is not to be denied. It may conceivably be due to chance that some of these accompanying conditions strike persons suffering from appendicitis, and yet some cases are highly suggestive of a common cause.

In reviewing the records appearing in the literature, one is constrained to wonder concerning the dietary habits of the families described. Only too often cases are recorded from hearsay or from the word of

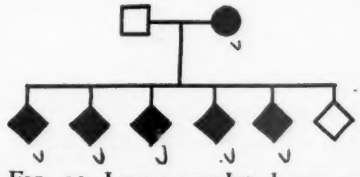

Fig. 12. Large angulated appendices. (Data from Van Meter.)

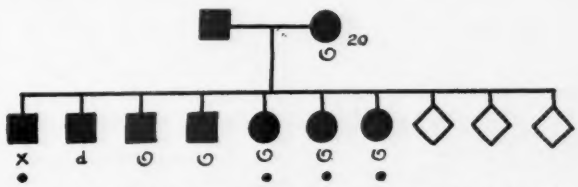

Fig. 13. Coiled appendices. (Data from Van Meter.)

Some of the pedigrees we have reviewed are so meager as to carry little weight, whereas others are quite convincing evidence that peculiarities or anomalies of the appendix are inherited as simple

[9] Cignozzi, O. Le appendiciti familiari. *Policlinico* sez. Chir., 29: 585–590, 1922.

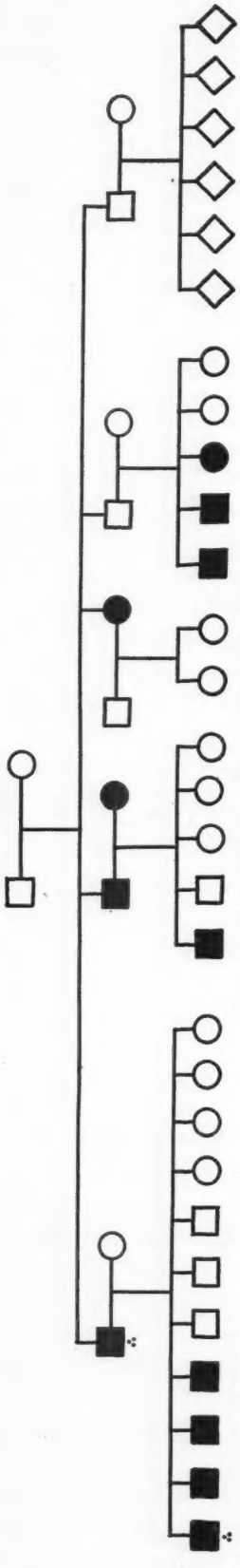

Fig. 14. Appendicitis. (Data from Magruder.)

a single patient. Had it been possible to follow the leads of some of these cases appearing in the literature, it seems probable that much more extensive pedigrees would have come to light, providing that the several diatheses for appendicitis are genetic and unit-dominants in nature, as they appear to be in the cases reported.

A pedigree of appendicitis which we now present is by far the most extensive one yet placed upon record as will be evident from an examination of Figure 20. Intimate acquaintance with this expanded network of relationships allows us to answer a number of criticisms which logically might be brought against the collection of appendicitis pedigrees from a genetic standpoint. The households represented are quite comfortably situated. The stock is primarily old Yankee. The intelligence and capability is high. Knowledge of balanced diets is complete. Nervous disorders are uncommon. Except in two cases, described as chronic by the surgeon, there has been no tendency to remove the appendix as a preventive measure, although tonsils have been excised as a matter of course. Hence the possible correlation between tonsillitis and appendicitis cannot be studied in this pedigree.

There have been some cousin marriages in the past in this family. The pedigree chart is a bit complicated due to the marriages of two brothers (F_{10} and F_{20}) to two sisters (F_{11} and F_{21}). This fact has necessitated two long parallel lines, each connecting a set of siblings.

Viewing the pedigree as a whole, one will note immediately that the cases are not distributed at random, as might be expected if no hereditary diathesis were present. On the contrary, in the upper section of the pedigree the cases occur sporadically in 6.5 per cent of the population, whereas in the lower section they occur in 35 per cent, or more than five times as frequently. This makes it apparent that some sort of hereditary diathesis is being transmitted in the lower portion of the pedigree.

The average age at the time of attacks shows a definite anticipation, the average ages being 52.5 years for the F_1; 32.3 years for the F_2, 13.4 years for the F_3. The one affected child in the F_4 was 7 years of age. It must be said, however, that F_1 11, although she had bouts of appendicitis for several years, was not operated on until the terminal attack from which she died at the age of 66.

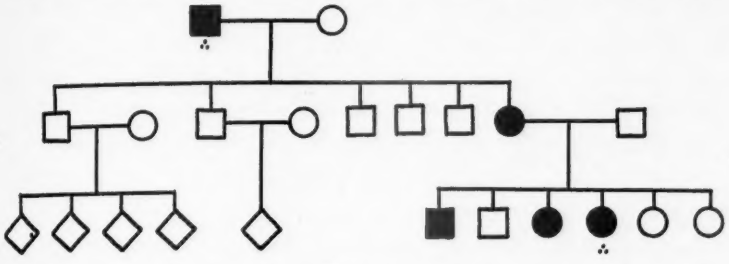

Fig. 15.

Fig. 16.

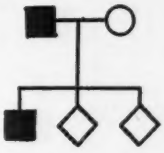

Fig. 17.

Figs. 15, 16 and 17. Appendicitis. (Data from Magruder.)

If we try to single out particular types of matings and apply Mendelian formulae, the data do not make sense.

There are sixteen affected males and twenty-three affected females, indicating that no male sex linkage exists. Eight matings between normals produced nineteen normals and thirteen affected, which is a wide deviation from the recessive Mendelian 3:1 ratio. Five families have one affected parent (not counting the last generation in which the children are still young). These families have eight normals and eleven affected, suggesting a unit-dominant.

In three families both parents have had appendicitis and in these, three children are normal and six are affected, also suggesting a unit-dominant. These data, with suggestions both of dominance and recessivity, cannot be reconciled with each other and the best we can say is that appendicitis tendencies are inherited in this family but in an irregular fashion.

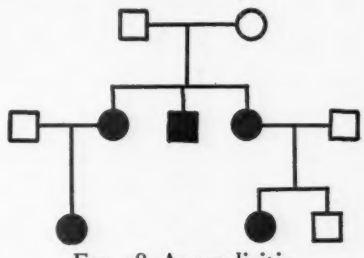

Fig. 18. Appendicitis.

If we study the twenty-one hospital records which we have been able to obtain, we find such diverse accompanying or predisposing factors as gall-bladder disease, coiled appendices, long appendices, retrocecal appendices.

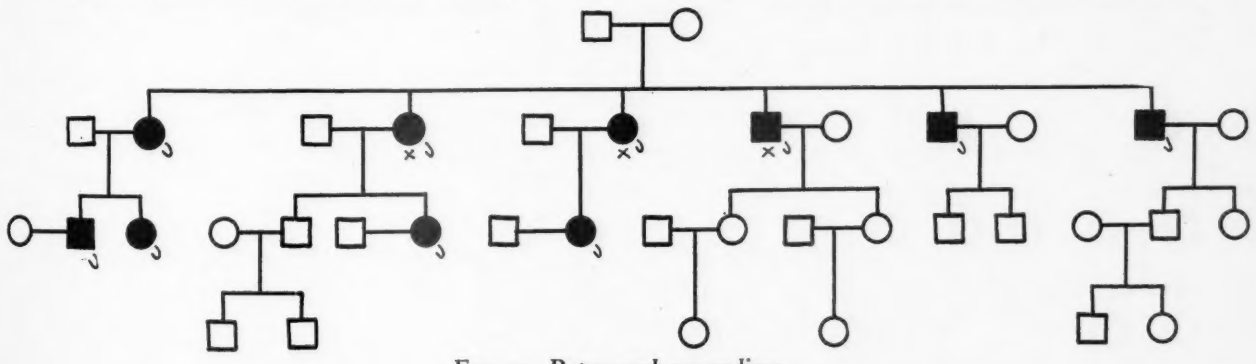

Fig. 19. Retrocecal appendices.

If we examine the portion of the pedigree where appendicitis is most concentrated we find that the complex consists of the appendicitis family of male F_2 27, who married into the appendicitis family of female F_2 28 (inclosed in dash line). F_2 27, his two sisters F_2 23 and F_2 41, as well as

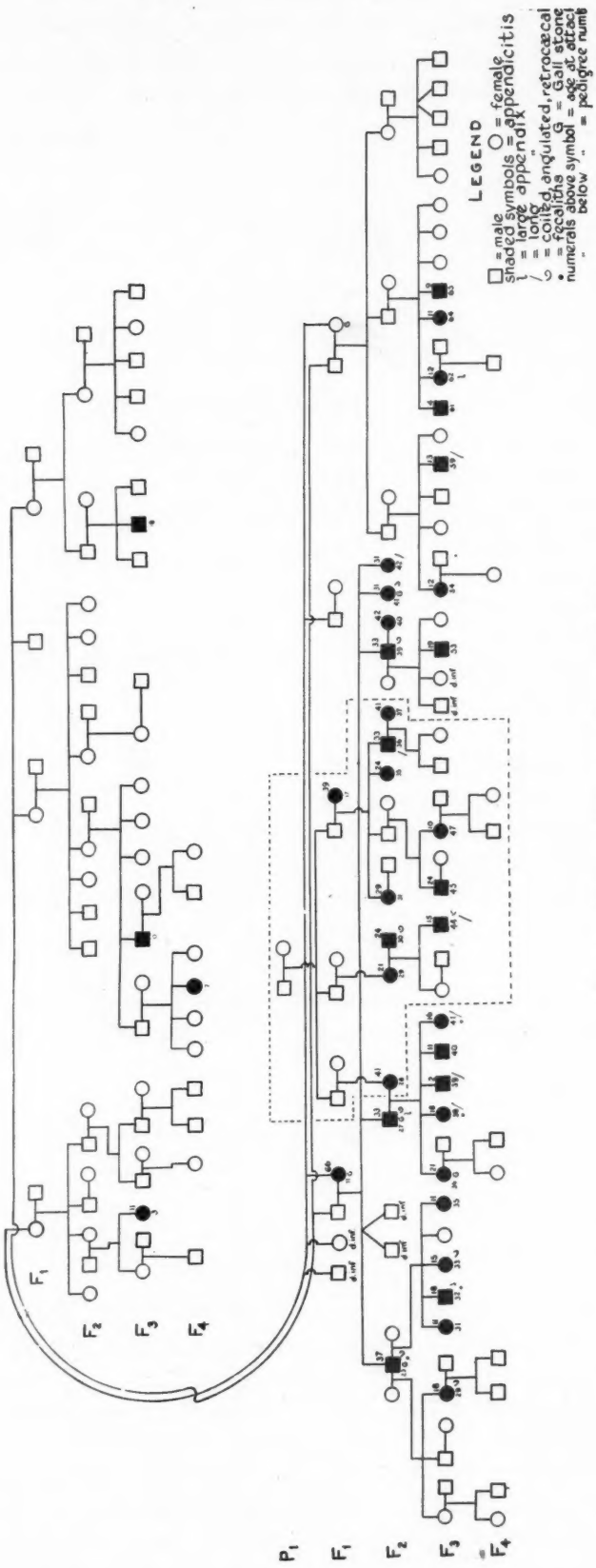

FIG. 20. Thirty-nine cases of appendicitis.

his mother and a maternal aunt, all had gallstones. In his wife's family no gallstones are recorded. His daughter (F_3 36) had gall-bladder trouble following her first pregnancy but she had no gall-bladder operation.

F_2 27 and three of his siblings (F_2 23, 39, 41) had coiled, kinked, or retrocecal appendices. His appendix, as described in the operation report, was large, and that of his sister F_2 42 was very long. F_2 27 apparently transmitted his "large" appendix to his three children (F_3 38, 39, 41) whose appendices were "elongated," "10 inches long," and "8 inches long" respectively. F_2 23 (the brother of F_2 27) who had a "sharp angle or twist" had, by two wives, five affected children (F_3 29, 31, 32, 33, 35), three of whom had appendices described as "angulated," "retrocecal," and "moderately kinked on itself." F_2 23, his child (F_3 32), and his two nieces (F_3 38, 41) all had fecaliths. The relatives, F_3 59 and F_3 62, had "long" and "large" appendices respectively. Thus, in this one family we have the predisposing factors of lithiasis, gallstones, long or large appendices, and bent, coiled or retrocecal appendices appearing as hereditary characters. (It is to be understood, however, that kinking or coiling of the appendix may frequently be acquired rather than hereditary.)

In the family of F_2 28 a father (F_2 30) and his son (F_3 44) have coiled appendices. Long appendices are recorded for F_2 36 and F_3 44.

Those findings are quite in contrast to our pedigree shown in Figure 19 in which the one predisposing factor of retrocecal form of the appendix is inherited as a simple, dominant unit-character, once appendicitis has appeared. To us it seems highly improbable that all the predisposing factors shown in Figure 20 could be different manifestations of one hereditary cause.

We are forced to conclude in view of all the facts recorded in the literature and from our own studies that there are a number of hereditary factors predisposing to appendicitis (some of which factors are inherited as irregular dominants), and that the genes for several of these predisposing physical factors, as shown in Figure 20, are concentrated in the germ plasm of F_1 11 and her descendants, one of whom (F_2 27) married into the appendicitis family of F_2 28.

SUMMARY

A review of the literature shows that a number of diverse hereditary constitutions are probably conducive to appendicitis. We present a pedigree containing thirty-nine individuals who have suffered from appendicitis. The tendency to appendicitis is hereditary in this pedigree, and seems to involve the transmission of several different types of predisposition.

The authors are deeply indebted to a number of hospitals and physicians who so kindly provided us with case histories and especially to Dr. John E. Ruisi of Westerly, R. I., who operated upon several of the patients and who has greatly assisted us with the literature.

A CLASSIFICATION OF FOREARM FRACTURES NEAR THE WRIST

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THE subject of this discussion is an anatomic and roentgenologic study of fractures of the forearm bones near the wrist, presented for the purpose of introducing a classification, with a system of internal measurement derived from anatomic landmarks. The classification, which has been observed and applied to cases for the past five years, and used for teaching purposes for the last two, is submitted for critical review and confirmation by other workers interested in these fractures.

FIG. 1. Posterior view of the hand in dorsal flexion and palm down position.

The late A. P. C. Ashhurst[1] wrote: "It is perhaps unnecessary to argue the desirability of classification; for without classification, the relation of one lesion to another can be neither remembered nor understood in any department of knowledge; and comprehension is a prerequisite for intelligent memory and for rational diagnosis and treatment." These classic words render further discussion of the value of classification superfluous, except to note that classification is not a simple listing of different types of lesions, in this case, fractures. Such a listing is a catalog but not a classification. A true classification should demonstrate some fundamental relation of one lesion to every other lesion.

FIG. 2. Palmaris longus tendon retracted with a portion of the palmar fascia attached.

The primary observation of this paper is that in suprastyloid fractures of the radius, the distance from the center of the ulnar

styloid base to the second carpometacarpal articulation equals the distance from the ulnar styloid base to the point where the fracture line joins the lateral surface of the radius. The exceptions are few. This observation is grounded in the three following concepts of applied anatomy, not greatly emphasized heretofore. The first two were mentioned in the December, 1937 issue of this journal.[2]

1. The proximal row of carpal bones move as a unit in transmitting force from the hand to the radius.

2. The distal row of carpal bones, except the greater multangular, combined with the four medial metacarpals, forms the central unit of the hand, in which any force entering the radius from the hand originates.

3. The recognition of the fact that the distal end of the ulna lags behind the radius in growth from childhood to adult life.

The problem toward which this work is directed is twofold:

1. When an individual falls on the hand, why does the site of fracture move distally in the radius as age increases, until adult life is reached?

2. What is the precise level of supra-styloid (Colles') fracture?

In Colles' original description, he placed the fracture line 1½ inches above the carpal extremity of the radius. Since that time, there has been a difference of opinion regarding the exact level of the fracture line. Eliason and his associates[3] refer to the confusion existing and place the fracture ½ to ¾ inch above the joint surface of the radius, and designate the lesion as "supra-styloid." Any review of the literature will show that attempts at the precise definition of this lesion, not excepting Colles' original description, have all been expressed in terms of external linear measurement, i.e., not in relation to regional anatomic landmarks.

The observations presented here are made in the usual anteroposterior films of the wrist in 180 degrees extension. The hand may be in midposition, radial or ulnar deviation. Under these conditions, one point of a compass is placed in the middle of the base of the ulnar styloid, and

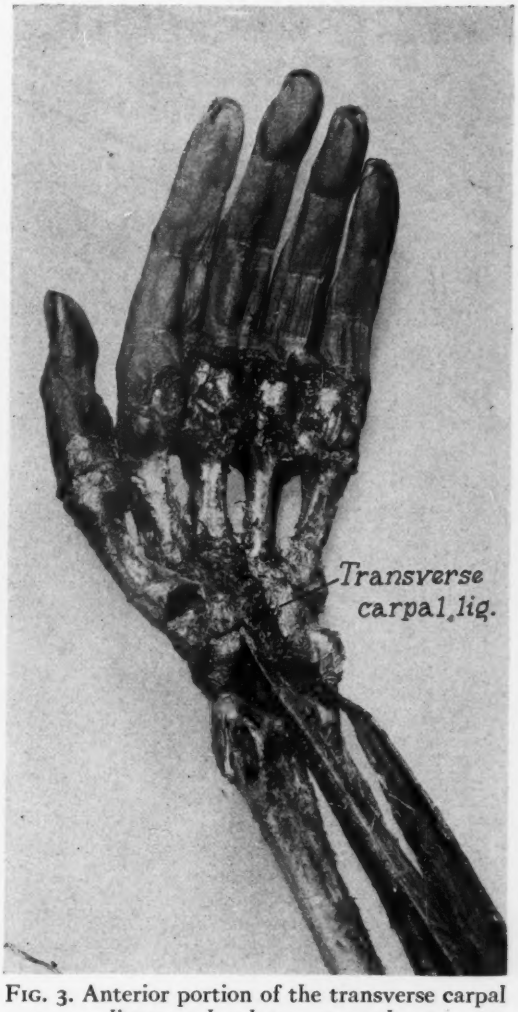

Fig. 3. Anterior portion of the transverse carpal ligament has been removed.

the other in the middle of the articulation between the os capitate and the proximal end of the second metacarpal. (Figs. 12 and 13.) With this distance as a radius and with the point on the ulnar styloid as a center, a line is drawn proximally, forming the arc of a circle which crosses the radius and ulna. This line will be found to intersect the fracture line where it meets the lateral border of the radius. (Plates I, II and III.) The circle with the term "circle test" is used in what follows, merely as a simple device to demonstrate that the middle of the proximal end of the central unit of the hand is the same distance from the

ulnar styloid, as the ulnar styloid is from the point on the radius where the fracture line joins the lateral border of the bone.

(*b*) An overlooked greenstick fracture in the lower end of the ulna, with resultant bending of the distal end of the ulna toward the radius. This is especially common in very young children.

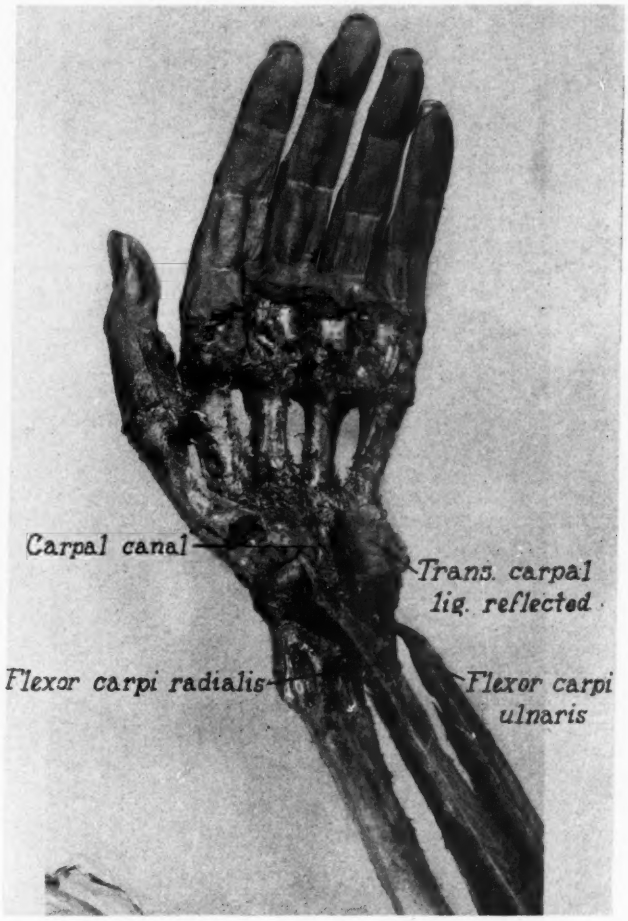

FIG. 4. Transverse carpal ligament severed from its attachment on the radial side.

Sources of error in the use of this type of measurement are:

1. The film should be a true anteroposterior film, i.e., under optimum conditions the full width of the distal radio-ulnar articulation should be shown. After one has applied the above maneuver to a hundred or more fractures of all ages in this region, and satisfied himself of the validity of the test, a moderate amount of distortion is readily discounted.

2. Any condition which changes the normal distance from the base of the ulnar styloid to the second carpometacarpal joint:

(*a*) A shortening of the carpus, such as that seen in severe arthritis with destruction of the cartilage.

(*c*) Fracture with displacement of the entire lower end of the ulna.

(*d*) An abnormally long or short ulna, or ulnar styloid.

Unfortunately, the simplicity and ease with which the test is applied may cause it to be regarded as an act of prestidigitation. In an effort to dispel this attitude, certain questions are anticipated.

1. *Of what use is the circle test?*

(*a*) It differentiates the typical fracture, sustained in a fall to the standing level, from the atypical fracture. The typical fracture is on the line or slightly distal to it, but is not proximal to it.

(*b*) It demonstrates that the suprastyloid fracture in children is governed by the same mechanics which operate in the production of the adult (Colles') type.

(*c*) It directs attention to growth changes in the distal end of the radius and ulna, identifying slight variations from the normal.

(*d*) It serves as a system of internal measurement based on anatomic landmarks, rather than on linear measurement.

(*e*) It defines suprastyloid (Colles') fracture of the radius as a double rotation (lateral and dorsal) fracture of the radius, in which the leverage effects of the central unit of the hand seem to be demonstrated.

2. *Why was the middle of the base of the ulnar styloid selected as the center of rotation?*

The ulna is more largely the continuation bone of the humerus, since it articulates with the humerus in a manner which permits motion in only one plane; whereas the radius not only moves in this same plane, but is permitted rotation on its long axis through the head and neck, while distal to the neck it rotates about the distal portion of the ulna. The distal ends of the radius and ulna are parallel members, firmly bound together by the strong ligaments of the distal radio-ulnar articulation, as well as by the interosseous membrane and the ligaments from the ulnar styloid to the carpus. The medial ligaments of the wrist are attached to the tip and as far proximally as the base of the ulnar styloid, while the triangular fibrocartilage is attached to its base.

In such an arrangement, if force is applied to the radius in its long axis from distally, and fracture of the distal end occurs, the fractured portion which is bound to the lower end of the ulna must rotate about a point in the lower end of the ulna as a center. If one wishes to state the proposition differently and show that the force comes from above or proximally, the result is the same. The lower end of the radius meets resistance coming through the carpus and is stopped and held, while the lower end of the ulna which does not enter into the wrist joint and which receives very little if any direct force, is carried on and has a tendency to ride distally past the lower end of the radius. The clinical proof of this lateral rotation of the distal fragment is seen in the lateral tilt of the fragment, together with the invariable displacement of the ulnar styloid fragment, when it is fractured, toward the carpus.

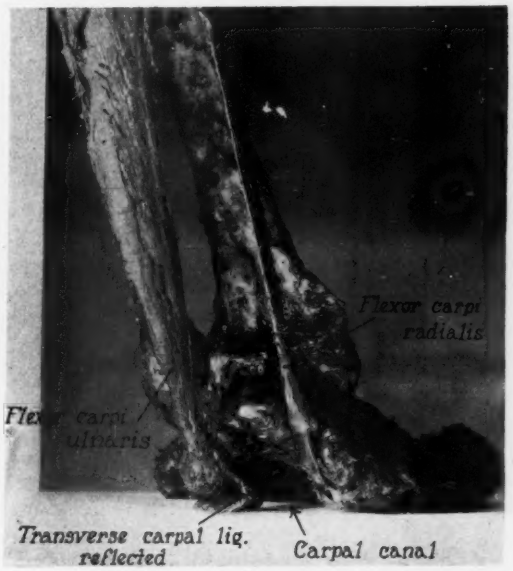

Fig. 5. Palm down position, showing the carpal arch which sustains the weight of the body in a fall on the hand.

3. *Why was a point chosen in the middle of the second carpo-metacarpal articulation?*

The selection of this point was not arbitrary. It rests on a conception of the functional anatomy of the hand and carpus, described in 1933,[4] and again more recently.[2] Even a cursory study of the anatomy of the distal end of the radius and ulna will show that the hand and carpus are rotated away from the ulnar styloid as a center, in a fall on the hand. But the task of demonstrating the use of the hand as a lever in guiding and disseminating force to the radius was found to be difficult and puzzling. It was learned that the proximal row of carpal bones act as a unit, moving as one piece and acting as an adjustable disc for the transmission of force from the remainder of the hand to the radius. (Figs. 12–15.) This conception of the function of the proximal row bones is substantiated by

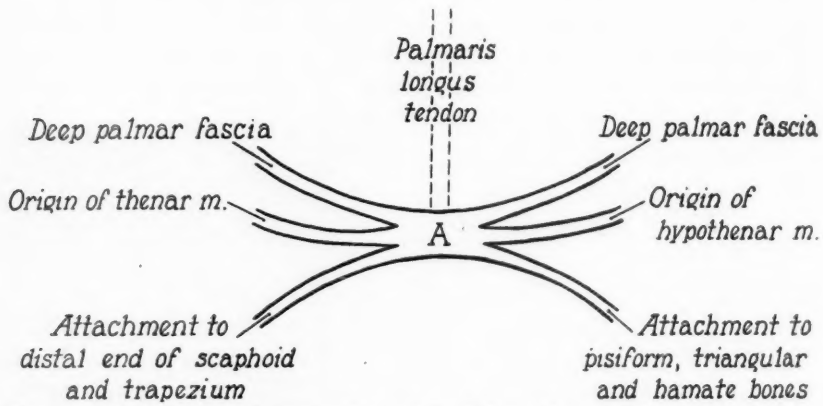
Palmaris longus tendon
Deep palmar fascia
Deep palmar fascia
Origin of thenar m.
Origin of hypothenar m.
A
Attachment to distal end of scaphoid and trapezium
Attachment to pisiform, triangular and hamate bones

FIG. 6. The fusion of the central portion of the palmar fascia (aponeurosis) with the trans. carp. lig., forms a structure of great clinical importance in any study of transmission of force through the hand, either to the carpal bones or to the radius. From a clinical viewpoint, it is a unified structure and is here labelled 'A,' the central tendon of the hand. The texts with which I am familiar do not properly interpret the functional significance of this central tendon. Its clinical importance is actually obscured when studied under classical anatomical subdivisions. It is quite apparent that any force received through the palm of the hand or through the hand used as a fist, is sustained in whole or in part by the central tendon.

The thick middle portion of the palmar fascia has two layers; an anterior whose fibers are longitudinal, and a deeper layer composed of transverse fibers which blend with the trans. carp. lig. The palmaris longus, an exceedingly variable muscle which is absent in 10–20 per cent of specimens, is a tensor not only of the palmar fascia but also of the entire structure of the central tendon. As seen in cross section, the central tendon is six tailed in three planes, having the palmar fascia in the anterior plane; giving origin to the thenar and hypothenar muscles in the middle plane; and finding attachment to the anterior surfaces of the bones which form the carpal canal, in a deeper and more dorsal plane.

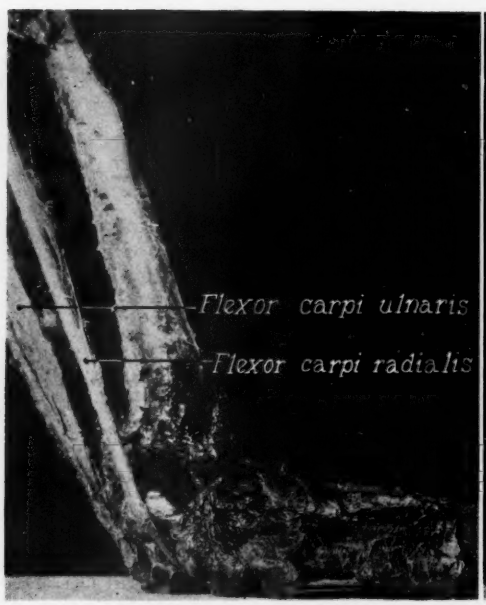

FIG. 7. Lateral view, dorsal flexion.

FIG. 8. Lateral view, extreme dorsal flexion.

the fact that none of these bones has muscles inserting on it. They are under control of the flexors carpi ulnaris and radialis through the connection with these muscles at either end of the proximal row, at the distal end of the scaphoid and through the sesamoid pisiform to the triangular. The extensor tendons on the dorsum of the wrist, both for the wrist and for the fingers, have no insertion on either the proximal or the distal row of carpal bones.

It should also be recognized that the distal carpal row bones, except the greater multangular, together with the four medial metacarpals, act as the central unit of the hand, between whose individual members there is scant movement. All the extensor tendons of the wrist and fingers insert in this central unit, except those to the thumb which go to the first metacarpal or distal to it. The carpal bones form an arch subtended by the strong transverse carpal ligament (Figs. 2–8, 11, 14) and it is this arch,

Fig. 9. A newborn wrist, anterior view.

Fig. 10. A newborn wrist, posterior view.

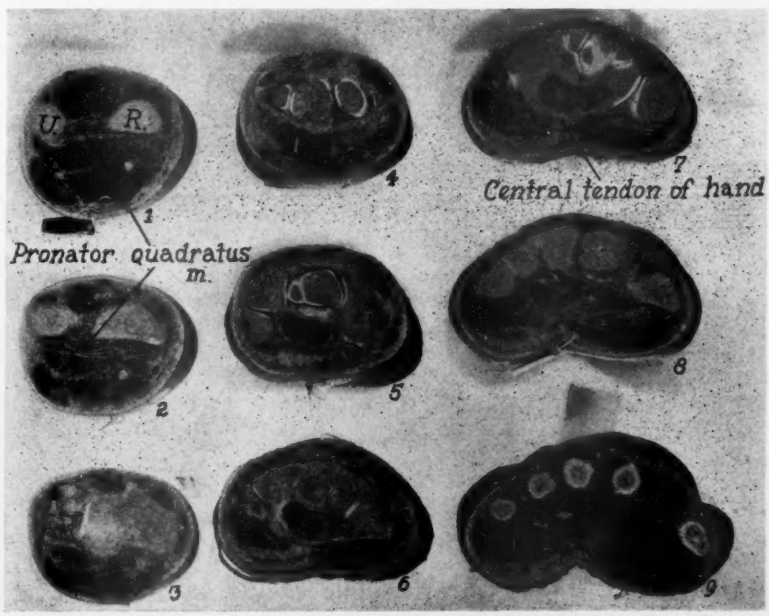

Fig. 11. Transverse sections through the wrist.

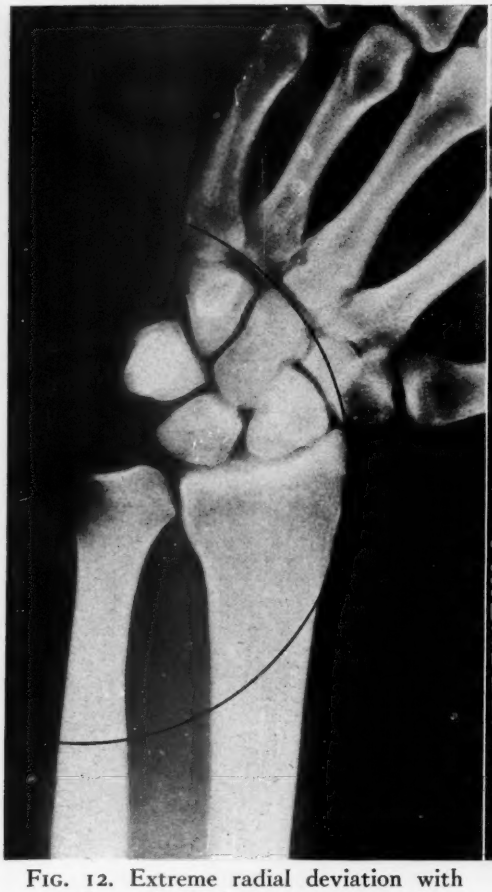

FIG. 12. Extreme radial deviation with circle applied. The third metacarpal, os capitate and semilunar bones are in line. The distal radio-ulnar articulation is shown in a direct a-p view. The tip of the radial styloid is at the midcarpal joint line. Note that the lateral styloid convexity of the radius is poorly developed.

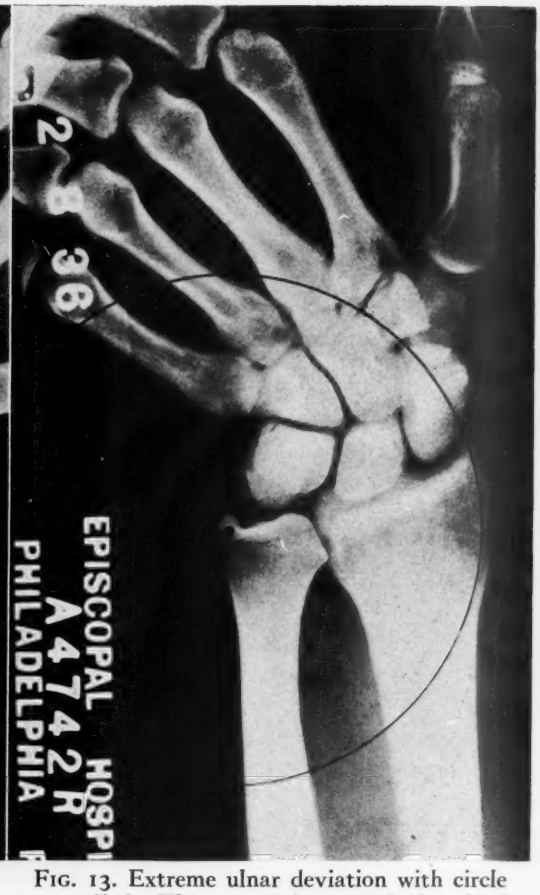

FIG. 13. Extreme ulnar deviation with circle applied. The os capitate and the third metacarpal are in line with each other but not with the semilunar. There is some rotation in taking the film in that a clear view of the cartilaginous area of the distal radio-ulnar articulation is obscured. The pisiform is slightly off the triangular in a proximal direction.

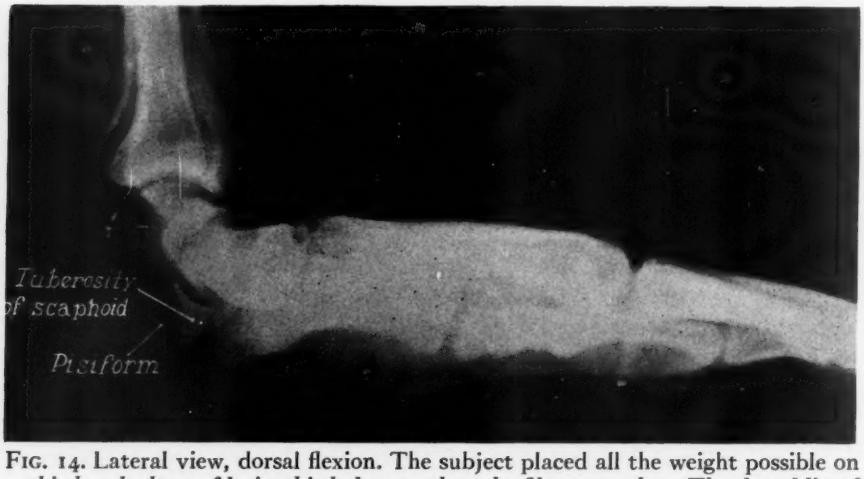

FIG. 14. Lateral view, dorsal flexion. The subject placed all the weight possible on this hand, short of losing his balance, when the film was taken. The dorsal lip of the radius overrides the proximal row of carpal bones. Note that the bones of the distal carpal row and the metacarpals are in the same horizontal plane.

with the central tendon of the hand, which sustains the full body weight in a fall on the hand. With these facts in mind, the puz-

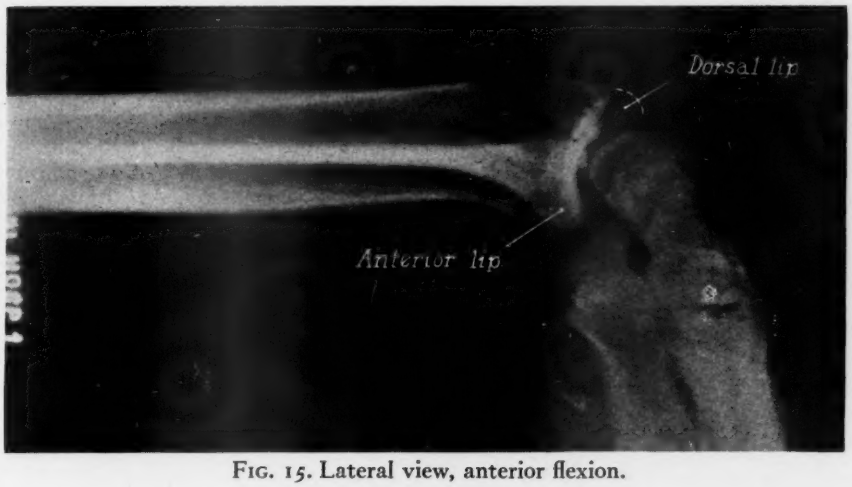

Fig. 15. Lateral view, anterior flexion.

zling action and distribution of forces in this region were somewhat clarified. After the proximal carpal row was recognized as an adjustable bony-cartilaginous plate, attention was focused on the central unit of the hand, consisting of the four medial metacarpals and the three medial carpal bones of the distal row.

Considerable interest was directed toward the os capitate from the beginning of this study, not only because it is the largest bone in the carpus, but also because it is the first bone to appear in the *x*-ray, casting a shadow at the age of six or eight months. It was assumed that of all the carpal bones, the capitate was subjected to maximal force from early life onward. Dissecting room studies also showed that the line drawn through the articulating surface between the os capitate and the second metacarpal, approximately bisected the chord which subtends the arc formed by the proximal surface of the distal carpal row. In other words, on the cadaver, this articulation represents the approximate midpoint of the proximal end of the central unit of the hand. The *x*-ray anatomy here, in the anteroposterior view, is very deceptive owing to the depth of the arch formed by the carpal bones. (Fig. 5.)

4. *Is it a coincidence that the circle formed by a radius whose length is equal to the distance between the ulnar styloid base and the middle of the second carpometacarpal joint, coincides with the lateral convexity of the adult radius?*

Early in this study, when such a distance was first used, it was so regarded. Obviously any fracture line which comes through to the lateral surface of the radius must be intercepted by the circle. Interest was heightened when films prepared with the hand in complete ulnar or radial deviation, showed that the second carpometacarpal articulation remained on the circle, in any position of ulnar or radial deviation or in midposition. (Figs. 12 and 13.) The matter rested here for a few months, when the interesting observation was made that in children, the circle passed through the line of fracture on the lateral surface of the radius with few exceptions, although it did not coincide with the lateral surface of the bone, but swung widely lateral to it. (Plate 1, Figs. 1, 2, 3, 5 and 6.) Before an ulnar styloid is formed, the compass is placed over the medial distal end of the ulna, where the ulnar styloid will later appear.

5. *How does the circle test for fractures of the radius hold true in both adult and childhood fractures, when the adult type is ½ to ¾ of an inch from the wrist joint, and the*

PLATE I. For legend see opposite page.

childhood type is 1 to 1½ inches from the joint?

The answer to this consists of one of the three main points supporting this classification. There is a simple anatomic fact which serves to place fractures in all age groups in this region in the same mechanical category. It is the observation that the distal end of the ulna lags behind that of the radius in growth, so that in the young child the distal end of the ulna is approximately ¼ to ½ inch proximal to the lower end of the radius. As age increases, the distal ulnar end tends to gain on the radius, until when growth is completed, it has reached its adult position. (Plate 1, Figs. 1–24.) The situation is reversed at the elbow where the growth of the ulna exceeds that of the radius. (Plate III, Fig. 72.)

6. *Is it true that the common site of adult fracture, where the cancellous bone of the distal end of the radius joins the cortical bone of the shaft, is a weak area in the radius?*

This area has been spoken of and taught as a zone of weakness in the bone, and it may be true, although no incontrovertible evidence was found in favor of or against this view. However, it is a fact, that at the level of the usual adult fracture line, the bone also changes its shape. The question then arose: Does the fracture line occur in its usual site, because of the change in

PLATE 1.

No. 1. M. S., age 20 months. Greenstick subperiosteal fracture of radius, distal third.

No. 2. F. K., age 20 months. Fracture of both bones in distal third.

No. 3. J. K., age 5. Fracture of both bones, distal third.

No. 4. J. K., age 7. Questionable epiphyseal separation. The distal end of the ulna is bent in toward the radius, causing the circle to miss the epiphyseal line.

No. 5. J. H., age 8. Fracture in distal third of both bones.

No. 6. B. S., age 8. Fracture in distal third of both bones.

No. 7. J. G., age 9. Epiphyseal separation of distal epiphysis of radius.

No. 8. W. W., age 9. Greenstick fracture in distal third of radius.

No. 9. A. H., age 10. Greenstick fracture in distal third of radius.

No. 10. G. R., age 11. Epiphyseal separation, distal radial epiphysis with fracture of distal end of ulna.

No. 11. G. R., age 11. Same patient as No. 10 but before reduction.

No. 12. G. G., age 11. Greenstick fracture distal third of radius. Circle misses the fracture line by almost a centimeter.

No. 13. J. M., age 12. Epiphyseal separation distal radial epiphysis. Circle misses by about 0.5 cm.

No. 14. H. B., age 13. Fracture of both bones in distal third.

No. 15. D. Y., age 13. Fracture of both bones in distal third with chip fracture of ulnar styloid.

No. 16. L. Z., age 13. Chip fracture of posterior border of the diaphysis, with probable reduced epiphyseal separation. Note how the ulna is bent in toward the radius. The circle misses by 0.5 cm.

No. 17. S. B., age 13. Chip fracture of posterior border of the dipahysis with epiphyseal separation and fracture of the ulnar styloid. Circle misses.

No. 18. H. W., age 13. Greenstick fracture in distal third of radius.

No. 19. R. M., age 13. Fracture of both bones in the distal third.

No. 20, W. G., age 14. Fracture of distal radial third with fracture of ulnar styloid. Circle misses slightly.

No. 21. N. S., age 15. Fracture of distal radial third with fracture of ulnar styloid.

No. 22. R. A., age 15. Fracture of distal radial third with fracture of ulnar styloid.

No. 23. J. C., age 16. Fracture of distal radial third.

No. 24. G. F., age 21. Subperiosteal fracture distal radial third. Circle misses slightly. Notice the distortion produced in taking the film, so that one sees the radial surface of the distal radio-ulnar articulation.

No. 25. M. C., age 17. Fracture distal end of radius with fracture of ulnar styloid. Circle intercepts fracture line. Epiphysis is closed, and distal end of ulna is somewhat atypical.

No. 26. F. K., age 14. Epiphyseal separation of the radius with fracture of the radial styloid.

No. 27. W. P., age 33. Fracture of radial styloid. No displacement. Cranking a car.

No. 28. W. D., age 25. Fracture of radial styloid. No displacement. Cranking a car.

No. 29. F. K., age 37. A comminuted fracture of distal end of radius. No displacement. Cranking a car.

No. 30. F. B., age 30. Fracture of radial styloid. No displacement. Cranking a car.

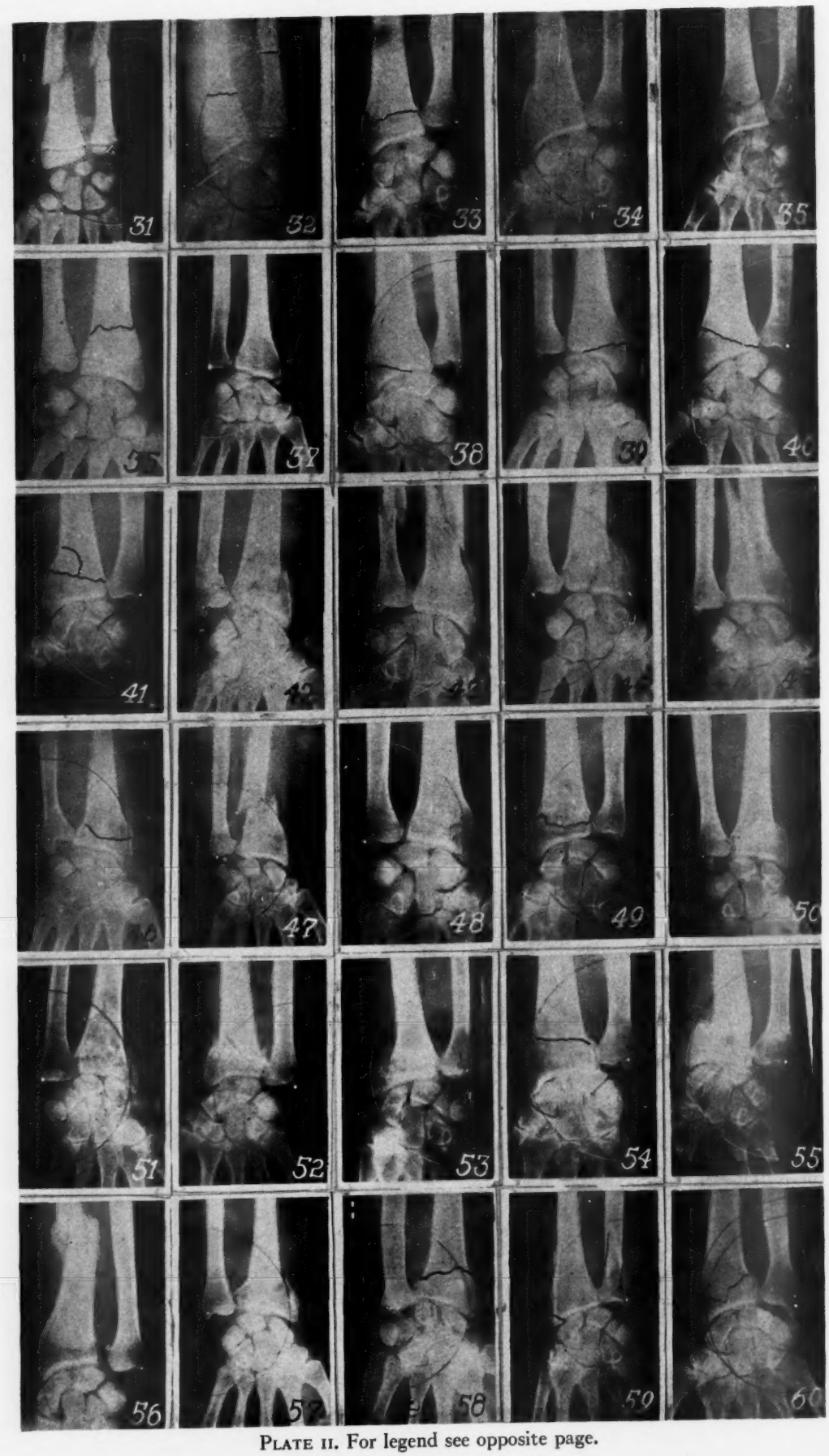

PLATE II. For legend see opposite page.

character or contour, or both; or is this an area in which the full effects of leverage forces generated in the hand are seen? The latter view was held to be the correct one. Cancellous bone is probably as strong as cortical bone, in its proper rôle, but its function is different. It seems to act as an instrument for condensing or focusing forces into the smaller diameter occupied by cortical bone. Jansen[5] wrote: "Summing up what previous chapters have taught concerning the structure of cancellous tissue, we come to the following conception: the lines along which pressure stresses

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Plate II.

No. 31. W. H., age 9. Fracture of both bones in the distal third. Cranking a car. Atypical.

No. 32. E. W., age 13. Fracture of both bones in the distal third. Cranking a car. Circle passes through fracture line.

No. 33. H. B., age 35. Fracture of distal end of radius with ulnar styloid. Cranking a car.

No. 34. J. B., age 44. Comminuted fracture of distal end of radius with ulnar styloid fracture. Cranking a car.

No. 35. S. P., age 24. Fracture in distal end of radius with ulnar styloid fracture. Cranking a car.

No. 36. N. B., age 18. Fracture in the distal end of radius with ulnar styloid fracture. Cranking a car.

No. 37. D. P., age 42. Fracture of radial styloid. No displacement. Fell down stairs. Resembles a Chauffeur's.

No. 38. H. B., age 32. Fracture of radial styloid. No displacement. Sustained in a fall. Resembles a Chauffeur's.

No. 39. J. Y., age 31. Fracture of radial styloid. No displacement. There is distortion present owing to the rotation of the bones when the film was taken. Fell from a ladder. Resembles a Chauffeur's.

No. 40. F. M., age 34. Fracture in the distal end of the radius. No displacement. Fell off a truck. Resembles a Chauffeur's.

No. 41. I. Y., age 62. Comminuted fracture in distal end of radius with fracture of ulnar styloid. Fell down steps.

No. 42. E. F., age 77. Fracture of both bones in the distal end.

No. 43. L. N., age 53. Fracture of both bones in the distal third. In auto accident.

No. 44. J. G., age 46. Comminuted fracture of the distal end of radius. Unusual violence. Fell from a ladder.

No. 45. M. M., age 35. Comminuted fracture of the distal end of the radius.

No. 46. E. B., age 40. Impacted fracture distal end of radius. Circle passes through fracture line, although ulna is slightly short, with an unusually large styloid.

No. 47. J. D., age 35. Fracture in distal end of radius with fracture of ulnar styloid. Unusual violence. Fell down stairs.

No. 48. A. M., age 41. Fracture in distal end of radius. Circle misses by 0.5 cm., but note the rotation of the radius in taking the film. Such a fracture will fall on the line in a direct a-p view.

No. 49. S. I., age 49. Fracture in the distal end of radius with fracture of the ulnar styloid.

No. 50. J. M., age 30. Comminuted fracture in the distal end of radius with ulnar styloid fracture.

No. 51. J. N., age 63. Fracture in the distal end of radius. Note the ulnar styloid shadow in the middle of the distal end of the ulna. The compass point is placed at the usual site on the ulna, ignoring the rotation of the ulna when the film was taken.

No. 52. E. S., age 31. Fracture in the distal end of radius with ulnar styloid fracture.

No. 53. M. M., age 43. Fracture in distal end of both bones.

No. 54. C. D., age 65. Fracture in distal end of radius with ulnar styloid fracture.

No. 55. M. F., age 42. Fracture in distal end of both bones.

No. 56. F. D., age 26. Fracture in distal third with callus formation. There is some rotation in taking the film, so that the circle misses the convex flare of the radius. The fracture is a true atypical fracture, in that it shows a fracture of the ulnar styloid associated with a fracture in the radius at an unusual level, well proximal to the circle. Fell while intoxicated.

No. 57. G. S., age 38. Comminuted fracture in distal end of radius. Fell down steps.

No. 58. H. L., age 49. Fracture in distal end of radius with ulnar styloid fracture. There is distortion of the film because of rotation, and an unusually long ulna. Circle misses by 0.5 cm.

No. 59. A. S., age 48. Fracture of the radial styloid and a spiral fracture in the distal end of the ulna.

No. 60. F. S., age 34. Fracture in distal end of right radius. Circle misses slightly. Note the unusually long and peculiarly shaped distal end of the ulna. Slight rotation in taking the film.

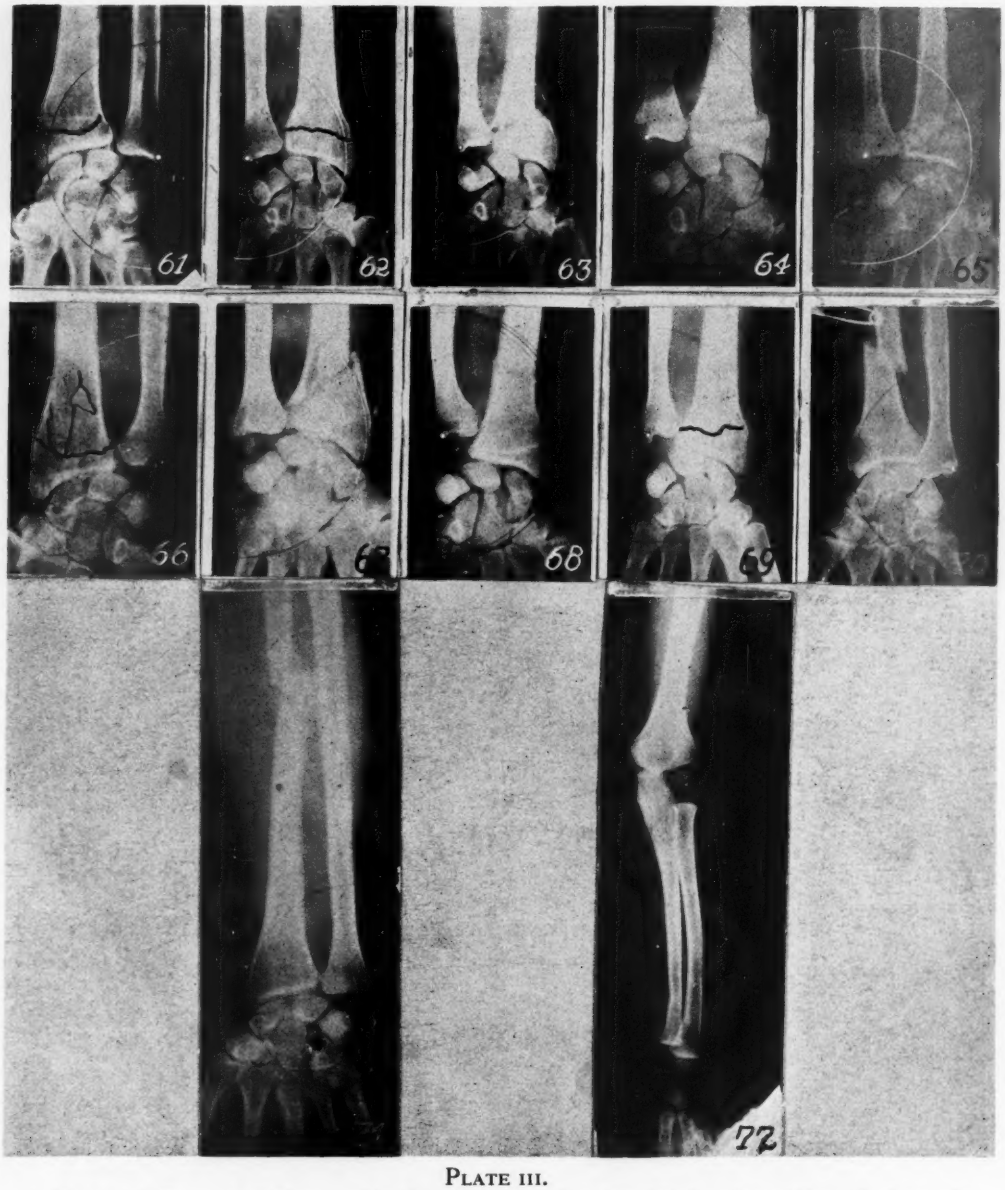

PLATE III.

No. 61. A. B., age 46. Fracture in the distal end of radius. Circle misses. Note the long ulna. In this case, the os capitate was very much shortened, because of old fracture or disease, and the case was the only one like it seen in the last five years. The condition in the os capitate was not acute, or of recent origin and was undiagnosed.

No. 62. A. H., age 52. Fracture in the distal end of radius with ulnar styloid fracture. Circle misses, owing to rotation in taking the film.

No. 63. C. B., age 39. Comminuted fracture in the distal end of radius. Fell down steps. Note that the use of the circle determines the amount and direction of displacement at a glance. It may pass through the end of the fracture line on either fragment, depending upon which fragment is displaced out of line with the hand.

No. 64. A. R., age 49. Fracture in the distal end of the radius and ulnar styloid fracture. The circle here passes through the end of the fracture line on the distal fragment. The medial displacement of the ulnar styloid fragment in this case was the only one seen and is included for that reason. Unusual violence. Fell from a ladder.

No. 65. A. K., age 35. Comminuted fracture in the distal end of radius. Unusual violence. Fell down steps.

No. 66. J. R., age 60. Comminuted fracture in the distal end of radius with ulnar styloid fracture. Unusual violence. Fell down steps.

For remainder of legend see opposite page.

run through bones, are represented by fasciculi of bone elements which fuse whenever the pressure lines are close together, but leave spaces between them whenever the pressure lines pass through relatively large transverse areas—as in joint surfaces. In each position of the joint surfaces a series of pressure lines passes from one bone to the other. So in the first place the number of these positions, i.e., the mutual mobility of the joint surfaces determines the texture of cancellous tissue."

It seems reasonable to believe that a fracture will always occur in the area of greatest stress. The fracture line occurs in a more proximal position in children, through cortical bone. Presumably this area of the shaft in a child's radius is as strong as other portions of the same bone.

7. *Why do patients in the years from 8 to 16 sustain epiphyseal separations?*

It is true that radial epiphyseal separations in the majority of cases tend to occur between these years, and this condition has been taught as an entity, unrelated to other lesions. If one observes the fracture line in different age groups, he will find the younger the patient, the greater will be the distance of the fracture line from the wrist joint (1 to 1½ inches). (Plate 1, Figs. 1–24.) As age increases, the fracture line approaches the wrist joint, until in the adult it is usually ½ to ¾ inch from the joint. As the center of rotation in the distal ulnar end moves distally, and the fracture line tends to fall closer to the wrist joint, a period of growth arrives in the years from 8 to 16, in which the zone of greatest stress coincides with the distal radial epiphysis. It appears that the area of greatest stress in the radius moves distally with the distal growth of the ulnar styloid area. That the epiphyseal line is not an especially weak area, prone to separation with fracture of its margins, is amply demonstrated by the fact that for the years prior to the age of 8 or 9, fracture may occur proximally while the epiphysis escapes injury. (Plate 1, Figs. 3, 5, 6, 14, etc.)

8. *In relation to the mechanics of fracture production, does the theory of ligamentous action in tearing off the distal end of the radius find support in this study?*

No. If the theory of ligamentous action is accepted in the mechanism of fracture production, it is necessary with the same theory to explain: (1) the impacted fracture; (2) the comminuted fracture with a V or T line extending into the articular surface; and (3) the suprastyloid fracture in children, in which the fracture is above the attachment of the ligaments. Also, if the ligamentous theory is accepted, it must be assumed that the ligaments on the flexor surface of the wrist are always stretched to their limits in a fall on the hand. That is to say, that an individual always falls on a hand held in extreme dorsiflexion. This was thought improbable.

It seems more logical to believe that ligaments about a joint come into play only when the muscles which control the movements in the joint have been ruptured or stretched to their utmost. In the case of muscles stretched to their maximum length, they may then act as ligaments across the joint line.

T and V fractures into the joint surface are thought by some to represent the result of greater force. This is reasonable, but raises the question of why we should

No. 67. M. G., age 38. Comminuted fracture in the distal end of radius with ulnar styloid fracture. Unusual violence. Fell from a stepladder.

No. 68. J. K., age 17. Fracture in distal third of radius with longitudinal fracture of the ulna. Note how the bending of the ulna toward the radius causes the circle to miss the fracture line. This arm was caught in a high speed lathe.

No. 69. A. O., age 37. Fracture in the distal end of radius. Circle misses slightly. Note the short ulna.

No. 70. E. R., age 52. Fracture in the distal third of radius. Circle misses. Atypical.

No. 71. J. J., age 22. Fracture in the middle third of radius with ulnar styloid fracture. Circle misses. Atypical.

No. 72. E. A., age 4. Full length view of the forearm bones.

observe this type of comminution, rather than another fracture more proximal in the same bone or in the humerus. The suggestion is offered, that with extreme violence, the comminution is the result of complete utilization of the protective articular mechanisms of the body, such as joint rotation. The T or V comminution is a spiral or oblique fracture of the fragment, and would seem to indicate a torsion force acting on the fragment.

9. *Are growth changes in the distal end of the radius and ulna of any importance in this study?*

Growth changes here are considered very important and are briefly described, as well as illustrated in the accompanying figures. (Plate 1, Figs. 1–24.) These changes are also clearly shown in the monograph by Eliason et al.[3] Before the age of 6 or 7 months, the carpus as recorded by the *x*-ray is devoid of any ossification center. The hand at this age and for the first few years of life is a flexible flipper hand, in which the cartilage component is much higher than in that of the adult. It may be assumed that this cartilaginous hand and carpus do not function as a firm unit for leverage in quite the same manner as do the more rigid bony hand and carpus of the adult. The ratio of the length of the hand and carpus to the length of the forearm is changed by growth to adult life. One newborn wrist (Figs. 9 and 10) was measured, and the distance from the tip of the third finger to the radiocarpal joint was equal to the length of the forearm when measured from the radiocarpal joint to the anterior flexion crease of the elbow. A few adult measurements made from the proximal volar flexion crease at the wrist to the tip of the third finger in one direction, and to the flexion crease of the elbow proximally, demonstrated an approximate ratio of seven for the combined length of the hand and carpus to nine for the length of the forearm. The changing ratio of length, as well as the flipper-type hand of the child, are factors to be considered in a fracture study of the forearm bones at this age. This newborn wrist was dissected primarily to learn whether or not the anatomic relationships about the medial ligaments of the wrist, and the attachment of the triangular fibrocartilage, were the same as in the adult. They were found the same.

Approximately at 6 months the ossification center for the os capitate begins to appear. At this age, there is no epiphyseal plate for the radius or ulna. Both bones are straight, with a slight concave flare at the distal end, on either side of each bone. It is especially important to notice that the level of the distal end of the ulna is from ¼ to ½ inch proximal to that of the radius. At 18 or 20 months, the radial epiphyseal plate becomes visible. The ulna is still short. Centers of ossification for the os capitate and hamate bones are visible. At three years, the ossification centers for the capitate, hamate and triangular bones are present, while the ulna remains short. At 7 or 8 years, all bones are present in the carpus and the epiphyseal plates of the radius and ulna are present.

At this age, from 7 to 9 years, when all the bones of the carpus are shown, certain changes occur in the radius. It loses its slight concavity along the distal borders, and begins to show a bulging outward above the epiphyseal line. At the age of 14 or 15 years, the lateral side of the lower end of the radius is convex outward; while the lower articular surface of the radius has been drawn distally, owing to the more rapid growth of the radial styloid, so that the radial articular surface now faces toward the ulnar side of the carpus. It is an interesting fact that this developing lateral convexity of the distal end of the radius, as well as the directional change in the facing of the distal radial articular surface toward the ulnar side, are both concurrent with the development of the ulnar styloid. The distal end of the ulna is still found to be somewhat proximal to its final adult position in relation to the radius.

At the age of 17 to 19 years, the epiphysis in each bone has united, leaving both the radius and ulna with an outward convexity

at the distal end. The convexity of the radius above its styloid is the much more striking of the two. The ulna has now assumed its adult position in relation to the radius, in which the distal end of the ulna is at the level of the ulnar side of the distal articular surface of the radius.

There is danger of confusing cause and effect in seeking the answer to the question of whether or not these growth changes in the bone contour represent the effect of a continuous rotatory force, centered in the distal end of the ulna, and acting in the coronal as well as in the sagittal plane. In such a debatable field, it is sufficient to recognize these changes, and to stress the most important point of all; namely, that the growth changes in the two bones are concurrent. In the light of the above observations, it may be fairly stated that the shape of the radial styloid and the ulnar-faced curvature of the distal radial articular surface are admirably fashioned and buttressed to receive and sustain pressure forces from a carpus moving along the circumference of a circle, whose center is found in the styloid of the ulna.

10. *Why is the lateral view of the x-ray film in suprastyloid fracture ignored?*

It is unnecessary to discuss the lateral view of these fractures. The dorsal rotation of the fragment is obvious and well known, and lateral views are more difficult to read. If the lateral end of the fracture line inevitably falls in the radius at a point equidistant with the second carpometacarpal joint, from the base of the ulnar styloid, it will be equidistant by measurement in any plane.

11. *Does the circle, upon which the classification is based, intercept the junction of the fracture line and the lateral border of the radius exactly in every case?*

No. The circle may miss the fracture line a distance of 0.5 cm. or so in the occasional case. It is not strictly applicable to transverse fractures at the base of the radial styloid (chauffeur's), although this fracture falls within its circumference. This fact accentuates the value of the circle. The mechanics of a fracture by backfire force are probably different from those in a fall on the hand. Fracture of the radial styloid base is a typical fracture for backfire force, but it is atypical when received in a fall on the hand. The circle test is more apt to fall astray in epiphyseal separation than in any other condition, possibly because the entire distal end of the ulna may bend without fracturing in children, causing the actual center of rotation in the ulna to fall at some point proximal to the distal end. The application of the test to reversed Colles' found it true in some cases and not in others. A clue as to why it may or may not work in reversed Colles' may be seen in the fact that some are true reversed fractures, and others are anterior fracture dislocations of the wrist.

An incidental result of continued application of the circle test is a tendency to examine the films much more closely than heretofore. Small amounts of distortion by rotation in taking the film are noted. An occasional short ulna, or an adult radius in which the lateral convexity is poorly developed, are quickly noticed.

12. *In a subject of this importance, dealing with an area in which most fractures occur, would it not be wise to make a complete study of consecutive fractures in the forearm bones in all the different fracture sites?*

This was considered necessary in establishing the classification. The circle test, to be of any value in classifying these lesions into one mechanical group, must include all typical fractures and must exclude all atypical fractures. Ashhurst[1] wrote: "the more one studies the subject, the fewer exceptions he will find to the general laws of mechanics; and the more his experience increases, the easier it will become to recognize the variants from the typical lesions."

At the outset, the films of all fractures of the radius and ulna in the *x*-ray files of the Episcopal Hospital in 1931 and 1932 were studied. They are listed below.

1. Suprastyloid fracture of the radius, combined with ulnar styloid fracture..... 152

2. Suprastyloid fracture of the radius, minus fracture of the ulnar styloid..... 122
   (74 were 15 years of age or younger; 48 were over 15 years of age.)
3. Suprastyloid fracture of both bones near the wrist..... 65
   (Oldest was 15 years; youngest 15 months; majority were 3–10 years of age.)
   (Two of these, age 9 and 13, were sustained in cranking a car.)
4. Epiphyseal separations of the radius..... 30
   (1 age 8; 2 age 9; 3 age 10; 1 age 11; 4 age 12; 5 age 13; 5 age 14; 3 age 15; 3 age 16; 1 age 17; 1 age 18; 1 age 19.)
5. Fracture of the radial styloid..... 25
   (17 of these were sustained in cranking a car.)
6. Fracture of the ulnar styloid alone..... 7
   (Usually by direct violence.)
7. Reversed Colles' fracture..... 6
8. Fracture of both bones proximal to the suprastyloid area..... 146
9. Fracture of the radius alone, other than head, neck or distal third..... 16
   (In upper third 10; 4 age 9, 1 each at 10, 11, 12, 13, 16 and 27.)
   (In middle third 6; ages 5 (2), 6, 10, 11 and 17.)
10. Fracture of the ulna alone, aside from the styloid..... 18
    (11 of these were spiral or oblique in upper, middle and distal thirds.)
    (6 were step fractures and one was greenstick.)
11. Fracture of head and neck of radius..... 28
    (5 of these were associated with posterior dislocation of the elbow.)
12. Fracture of the coronoid process..... 5
13. Fracture of the olecranon process..... 13

Fractures in the last two groups were often associated with other fractures about the elbow, often atypical. In order to gain a better idea of the ages of the epiphyseal separations, a second group of fifty-two was studied. The ages were: one age 2; one age 4; one age 7; two age 8; three age 9; three age 10; two age 11; thirteen age 12; nine age 13; nine age 14; four age 15; three age 16; one age 18.

The first four divisions are placed in one group by the circle test, and represent more than 50 per cent of all forearm fractures.

It was noted that oblique, spiral and so-called step fractures occurred in areas which are not protected by an adjacent joint permitting rotation. Fractures in bones near joints which permit free motion in more than one plane are commonly transverse fractures. Examples of this are seen in the transverse fracture of the surgical and anatomic necks of the humerus and the femur; in the supracondylar fracture near the elbow, where with the hand fixed, the extremity may rotate on the head of the radius; and in the suprastyloid fracture of the radius. Aside from the styloid and olecranon, spiral fractures are typical for the ulna.

The conditions existing in the radius are unusual and a spiral or oblique fracture is atypical for this bone. The radius is so arranged that rotation for the entire bone is easily permitted, in that it is a parallel bone to the ulna, where the ulna represents the fixed portion of this arrangement. The wrist joint is a diarthrodial joint, allowing motion in two planes so freely as to resemble the free rotatory motion of an enarthrodial joint. This movement of the wrist joint is possible, however, only in 180 degrees extension, and becomes progressively less as one approaches complete dorsal flexion, in which position the carpus is fixed in the joint, and the movements of radial and ulnar deviation completely disappear. Dorsal flexion is the position of the hand in falling, and the movements of the wrist in this position are limited. One might question the rotation entering into this arrangement, which could protect the suprastyloid area from a twisting force. The answer is found in the fact that the ulna is free to rotate on its own axis in the distal radio-ulnar joint, and with the humerus, it rotates around the head of the radius when the elbow is extended and the hand is fixed.

Spiral and oblique fractures, which may be assumed to represent the result of torsion forces, are commonly found in the middle of the shafts of the long bones, as in the humerus, the ulna where the spiral and so-called "step" fractures are the rule, and in the middle and distal thirds of the tibia where, with the knee in the extended weight-bearing position, there is an absence of any protective rotatory joint motion. They are also seen in the distal fragment in

the T or V comminuted fracture of the suprastyloid area.

The films pictured in the three plates were selected to show the less accurate applications of the circle test and the reasons therefore. Some cases showing its exact application are interspersed. In order to show the growth changes, the figures in Plate I, Figures 1 to 24 are arranged in the order of increasing age. These are routine films taken by Dr. R. S. Bromer, former roentgenologist to the Episcopal Hospital.

SUMMARY

The leverage effects of the hand and carpus in the production of suprastyloid fracture in the radius by a fall on the hand have been studied. Applied anatomy from original dissections; an analysis of all types of fractures of forearm bones for a two year period; and a series of *x*-ray films with illustrative circles drawn, have been presented in an effort to correlate the applied anatomy of the region with the known incidence and location of fracture as recorded by *x*-ray. A classification based upon a circle test used in anteroposterior films is submitted.

CONCLUSIONS

The location of the fracture line in the distal end of the radius, sustained in a fall on the hand, bears an unchanging relationship to the bones of the carpus, irrespective of the patient's age. This relationship is demonstrable in a high enough percentage of cases to remove it definitely from the realm of coincidence. It is suggested that epiphyseal separation in the distal end of the radius, is the result of increased stress occurring at that site during a certain age period, and not the result of an assumed weakness of the epiphyseal line.

Anatomic material was provided by the unfailing generosity of Dr. E. R. Clark, Professor of Anatomy in the Medical Department of the University of Pennsylvania. I am indebted to Mr. B. B. Varian in the same department for the pictures of the dissections shown in all the figures, and for the film photographs shown in the plates. Dr. Lowry Allen, roentgenologist to the Episcopal Hospital, kindly made the films pictured in Figures 12, 13, 14 and 15. Lettering, numerals, and the diagrammatic sketch shown in Figure 6, are by Miss Edna Hill of the University of Pennsylvania Hospital.

REFERENCES

1. Ashhurst, A. P. C., and Bromer, R. S. Classification and mechanism of fractures of the leg bones involving the ankle. Based on a study of 300 cases from the Episcopal Hospital. *Arch. Surg.*, 4: 51 (Jan.) 1922.
2. Snodgrass, L. E. Fractures of the carpal bones. *Am. J. Surg.*, 38: 539 (Dec.) 1937.
3. Eliason, E. L., Goldsmith, R., and Pendergrass, E. P. Fractures of the Humerus, Radius and Ulna, p. 216. New York, 1925. D. Appleton and Company.
4. Snodgrass, L. E. End-results of carpal-scaphoid fractures. *Ann. Surg.*, 97: 209–216 (Feb.) 1933.
5. Jansen, M. On Bone Formation, Its Relation to Tension and Pressure. London & New York, 1920. Longmans, Green, University Press (Manchester).

ACROMIOCLAVICULAR AND STERNOCLAVICULAR JOINT INJURIES*

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THE clavicle is unique in that while acting as the principal support that keeps the shoulder girdle from collapsing upon the thoracic cage, the clavicular articulations at both its inner and outer ends mechanically are the most unstable joints in the body, being entirely dependent upon the ligamentous attachments for their integrity. That the acromioclavicular and sternoclavicular joints are not more often subluxated, is probably due to the great range of the glenohumeral and scapulothoracic movements. The one force to which the clavicular joints will yield most readily is a lateral thrust. Should the thrust be sudden, violent and directly from the side, dislocation of the sternal end of the clavicle is most likely to occur. When the lateral thrust has a caudad component, or particularly if it acts from the posterior aspect of the shoulder, the acromioclavicular joint bears the strain, and may yield with partial or complete dislocation. While fracture of the clavicle is common in childhood or adolescence, true acromioclavicular or sternoclavicular dislocation rarely occurs at these ages, and is almost always encountered in adults, in whom clavicular dislocations are far more frequent in relation to the number of clavicular fractures than in children.

According to the amount of force and the time through which it acts, injuries of the acromioclavicular joints may be divided into sprains, incomplete dislocations (subluxations) and complete dislocations of the joint. In the sprain, pain on motion of the arm, particularly on elevation or load lifting, tenderness at the joint, and local swelling are encountered. At times, abnormal motion, not found on the normal uninjured joint, is encountered. Naturally, the transition stage from sprain to separation is not great, and it is often difficult in the first few days to judge whether the injury is a severe sprain or partial subluxation, until the response to therapy is observed. There are no demonstrable changes found in the radiographs of such cases.

When greater force has acted, definite abnormal false motion is present, the tenderness and swelling of the joint is marked, and usually marked difficulty is experienced in the use of the injured limb. In *x*-rays of the shoulder, (anterior-posterior) taken with the patient sitting or standing, the outer end of the clavicle is found displaced upward from 2 to 4 mm. above its usual level in relation to the acromion. In such incomplete dislocations of the acromioclavicular joint, the superior and inferior acromioclavicular ligaments are torn, and not infrequently, there is evidence that the coracoclavicular ligaments may suffer damage of varying extent as well.

In complete acromioclavicular dislocation, attempted elevation of the injured arm increases the obvious deformity, and the clavicle may ride above and project beyond the medial articular border of the acromion. It is not fully recognized that posterior displacement of the clavicle in relation to the acromion is present in every case as well. By comparing the anterior-posterior radiographic projection (Fig. 1) with the lateral view of the same case, this wide separation is vividly portrayed. Such lateral roentgenograms may possibly be of value in determining the true degree of some of the severe "incomplete" acromioclavicular separations, for the roentgen-

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ologist's appreciation of the true clinical condition often is at variance with the physical signs found in the injured shoulder.

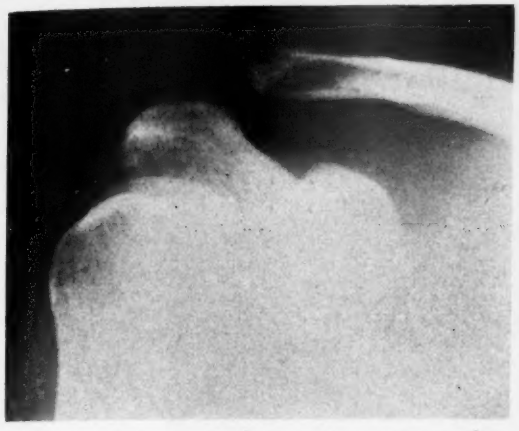


Fig. 1A. Anteroposterior roentgenogram taken with patient standing, showing a clinically complete acromioclavicular dislocation with tear of trapezoid and conoid coracoclavicular ligaments.

Sir Robert Jones[1] remarked of acromioclavicular dislocations; "Replacement is easy. Retention of the bones in position long enough to insure strong repair is almost entirely neglected by the majority of the profession." His form of treatment consisted in the use of the brachioclavicular sling, and apparently in his hands it was quite adequate. There are many modifications of this device, and all employ the general principle described by Jones. Inability to maintain the reduction, pressure phenomena about the tip of the olecranon, and probably unintelligent use of the method gradually led to the conception that complete acromioclavicular dislocations never healed without excess mobility, and that frequently a painful, unstable arm and shoulder were the result of the injury.

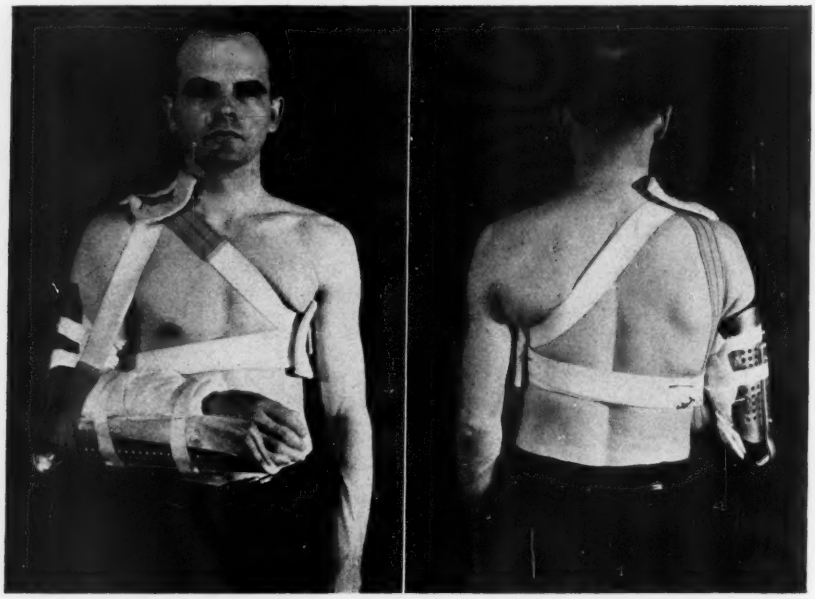
Operative methods varied from the use of a single wire suture, bone pegs, bone grafts, to the insertion of a long thin wood screw through the acromion, across the joint, into the clavicle. While at first glance, these efforts seemed to solve the problem, in practice, wires or pegs broke, screws went astray unless guided by a sharpshooter, and the essential point of restoration of joint function, *controlled motion*, was neglected. The acromioclavicular joint, a diarthrodial articulation, is constructed for motion.

Fig. 1B. Lateral roentgenogram of same shoulder, showing marked dislocation of clavicle backwards. The extent of separation cannot be appreciated in stereoroentgenograms.

One ordinarily thinks of shoulder motion taking place at the glenohumeral joint, and while recognition of concomitant movement is universal, the part played by acromioclavicular and sternoclavicular joints is often lost sight of. Mollier,[2] whose studies are not generally available, gives the range of motion in the joints as follows:

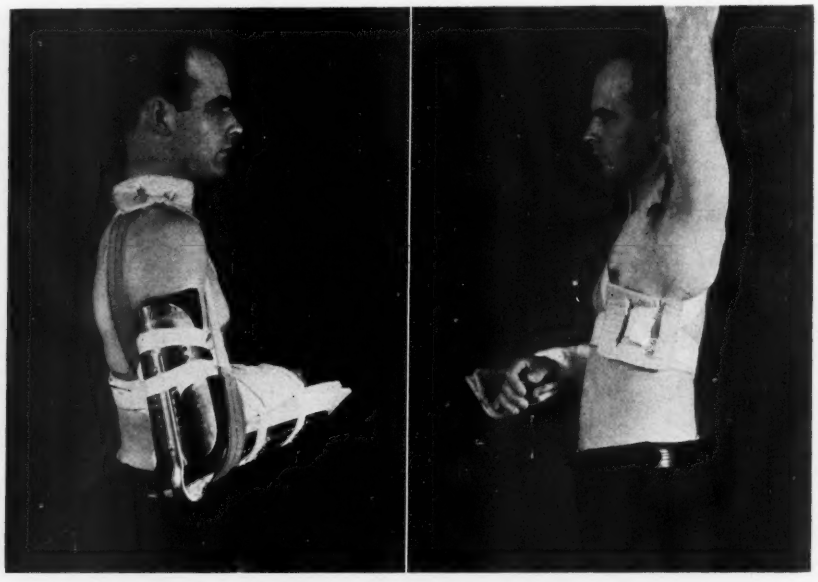
	Degrees
Acromioclavicular joint axes:	
Vertical	15
Sagittal	19
Frontal	29
Sternoclavicular joint axis:	
Vertical	37
Sagittal	20
Horizontal	22

He has demonstrated very clearly that for every change in position of the scapula, there is a corresponding change in the position of the clavicle reflected by movement at these two joints, and if either or both should be fixed and immobile, the total range of shoulder motion would be markedly impaired.



A B

FIG. 2. A, a metal right angled external elbow splint is well padded with saddler's felt. The patient's arm is heavily wrapped with cotton sheet wadding. The splint is applied to the arm with adhesive and gauze bandage. Then the acromioclavicular region is injected with 1 per cent novocaine, the dislocation of the joint reduced, and the elastic traction bands applied as shown. The traction bands are made of 2 inch cotton webbing, sewed to a central 2 foot strip of heavy 2 inch woven elastic webbing. B, back view. Note that the posterior vertical traction strap (gray striped) is carried beneath the encircling body strap. This holds the arm to the side securely. After two or three weeks in complete dislocations, the elastic vertical strap may be placed outside to allow beginning shoulder motion.



C D

FIG. 2. C and D, side views. Both strap crossings are anchored by slotted felt pads, which also ensure comfort.

Bunnell[3] realized the inadequacy of operative treatment by fixation, and stressed the fact that the tearing of the coracoclavicular ligaments was the essential feature in complete acromioclavicular dislocations. He devised a fascial suture plastic reconstruction of these structures as well as suture with fascia and silk of the acromioclavicular joint itself. Henry[4] of Minnesota had previously used such a method, and later described the operation. Ralph F. Bowers[5] reported six cases in which reconstruction of the ligamentous apparatus was carried out by means of heavy braided silk. The results of the operation in the hands of these surgeons were good. It cannot be said, however, that as a whole, the operative reconstruction of the ligaments has been followed by early return to work without residual disability.

Since 1932, in the Fracture Clinic of the Stanford Surgical Service, we have used a modification of the brachioclavicular sling which has proved satisfactory and adequate in the complete as well as the incomplete subluxations of the acromioclavicular joint. The effectiveness of the splint depends upon (1) immobilization of the elbow of the injured arm at a right angle, thus furnishing—a stable fulcrum from which effective upward traction of the arm and shoulder may be obtained; and (2) the use of elastic traction at the same time pressing downward upon the clavicle while the splinted arm, with its acromion, is pulled upward. The principles embodied in the splint are far from new, but the simplicity of the apparatus, and its effectiveness recommend its use. (Fig. 2.)

Certain suggestions may be added that may seem unnecessary, but in the light of teaching experience and practice deserve restatement. No splint on application reduces a fracture or dislocation by itself. The act of reduction must first be accomplished by the physician and then maintained by proper application of the apparatus. Novocaine (1 per cent, 10 to 15 c.c.) is usually sufficient for anesthesia and muscular relaxation. In these complete dislocations with the acromion riding forward under the clavicle, the operator's fist in the patient's axilla serves a sa fulcrum, and the patient's arm can be brought to the side of the body, levering the shoulder girdle and the acromion laterally from beneath the projecting clavicle. The flexed and splinted elbow is then pressed upward and backward, while the operator's opposite hand shoves the clavicle down and forward into its accustomed articulation with the acromion.

The dislocation reduced, the elastic and cotton webbing strap from the elbow is crossed over the injured shoulder, held in place above medially on the clavicle by slotted felt pads; crossed again in the opposite axilla (a similar slotted felt pad prevents chafing and "riding up" at this crossing) to encircle the body for final buckling or fastening. The slotted felt pads are important in that they insure the strap crossings' remaining in the desired position and prevent "creeping" as well as chafing of the skin.

For the first two weeks arm motion is prevented by passing the encircling body strap over the posterior vertical traction band of the arm. After a fortnight, the encircling body strap may be passed directly around the body, leaving both vertical arm traction straps free. The patient may then abduct, forward flex, and rotate the shoulder within a fairly wide range (70 degrees) without pain or recurrence of the dislocation, and so prevent shoulder stiffening and contracture. The splint should be used for four to five weeks in all complete separations, following which the usual muslin sling may be used until elbow and shoulder motions, encouraged by exercise, are quite free and comfortable.

Since 1932 we have treated thirty acromioclavicular joint injuries in the fracture clinic of Stanford Surgical Service and in private practice. The severity of the injuries and the end results are summarized in the accompanying charts. Naturally, sprains of the joint recover promptly, but as mentioned before, the gradation from

severe sprain to subluxation is not sharp, and the more disabling sprains recovered quickly with the use of the brachioclavicular traction splint. Relief of pain was noteworthy in every case in which the splint was used. Most of the patients had strapping, muslin slings or Velpeau's bandages when first seen. The relief is immediate and coincident with real support of the weight of the dependent arm. (Chart I.)

ACROMIO-CLAVICULAR SPRAIN

AGE	SEX	TREATMENT	DAYS OF DISABILITY
17 years	Male	Strapping	None (school boy)
21 years	Male	Sling	10 days
16 years	Male	Sling	6 days
20 years	Male	Sling	Not followed
33 years	Male	Sling	10 days
47 years	Male	Strapping and sling	8 days
21 years	Male	Strapping and sling	6 days
40 years	Male	Strapping and sling	21 days
33 years	Male	Brachio-clavicular splint.	6 days
22 years	Male	----------	Not followed

Average age—28 years. Average disability—8.3 days

CHART I.

The virtue of the splinting method is best shown in the ten complete acromioclavicular dislocations (Chart III). All of these patients have returned to active work, have been followed, and have remained free of pain, and of instability of the shoulder joint. (Fig. 3.)

An injury similar in nature and occasionaly accompanied by subluxation of the acromioclavicular joint is fracture of the outer end of the clavicle distal to the insertion of the coracoclavicular ligaments. Without tear of the coracoid and trapezoid ligaments the displacement of fragment is minimal and is easily treated, occasionally

ACROMIO-CLAVICULAR SEPARATIONS, INCOMPLETE.

AGE	SEX	TREATMENT	DAYS OF DISABILITY	PERMANENT DISABILITY
12 years	Male	16 days	16 days	None
45 years	Female	Sling 3 days – splint strap. 3 days 5 weeks	55 days	None—returned to work
27 years	Male	44 days no – splint treatment 3 weeks	65 days	None—returned to work
21 years	Male	Strapping – splint 9 days 21 days	34 days	None—returned to work
65 years	Male	Sling – splint 1 week 2 weeks	29 days	None—returned to work
26 years	Male	splint 20 days	56 days	None (also fracture of scapula)
33 years	Male	splint 32 days	48 days	None

Average Age—32.7 years. Average period of disability—43.2 days. Permanent disability—none.

CHART II.

In the incomplete separations or subluxations, the results were quite as satisfactory with an average of 43.2 days away from work; no permanent disability remained. (Chart II.)

with a simple sling. When the ligaments are torn, however, the displacement may be extreme. In such patients the brachioclavicular splint was effective in maintaining alignment and contact of the fragments. (Chart IV.) In one patient an uncoöperative alcoholic transient, no adequate reduction was obtained and no follow-up could be secured. This patient had a severe abrasion and contusion of the elbow. The application of the brachioclavicular elastic traction splint was followed by ulnar palsy. The one prolonged disability was in a patient who developed acute renal colic with calculus. He was referred to the genitourinary clinic and did not carry out the prescribed exercises. Prolonged stiffness of the shoulder was the result.

Sternoclavicular dislocations occur far less frequently than the separation at the outer end of the clavicle. With complete dislocations of this joint the extra-articular ligamentous structures suffer as well as the ligamentous portions of the joint capsule. Here the costoclavicular ligament ruptures and allows the clavicle to ride forward and across the jugular notch. In treatment of these infrequent injuries, operation has not gained the popularity that reconstructive surgery of the acromioclavicular joint enjoys. Nevertheless conservative treatment is often ineffective and results in laxity of the joint ligaments and recurring subluxation on heavy effort, which is rarely disabling to an extreme degree.

Those hopeful methods that attempt to maintain the medial bulbous clavicular end in its shallow articular facet by pressure pads over the joint are useless. They conceal the recurring dislocation beneath layer on layer of adhesive. The shoulder can be used as a fulcrum to good purpose, however, and by applying a well fitted clavicular cross, and by so pulling the shoulders and the clavicle up, out and back, one can maintain the sternoclavicular joint in its normal relations until the costoclavicular and articular ligaments are healed. The joint can be inspected at all times and the splint readjusted. At least four weeks of constant wearing of the cross splint are necessary to allow adult normal tensile strength to be developed in the torn ligamentous structures. Three patients treated in this manner recovered with no false motion or tendency to subluxation of the joint, and their average disability was fifty-four days. One of these patients suffered an oblique fracture of the inner end of the clavicle, splitting the joint surface, the subsequent displacement taking the form of the usual sternoclavicular dislocation. The reduction and maintenance of

Fig. 3. A, complete acromioclavicular dislocation with the clavicle overriding the acromion, while the entire shoulder girdle has dropped down and forward. One's fist in the patient's axilla serves as a fulcrum, the patient's arm serves as a lever, and adducting the elbow to the body forces the shoulder girdle outward to allow reduction of the dislocation. B, same joint twenty-seven months later. Clinically there is no deformity, no false motion, no pain and no limitation of motion as compared with the normal shoulder, in spite of mild arthritic changes in the joint.

position was carried out in the same manner as for a dislocation. The patient recovered with normal motion of the shoulder, but with excess callus at the site of injury. One may contrast treatment with the clavicular cross with other forms as shown by two patients in Chart IV. One was treated with an aeroplane abduction splint for six weeks, given a sling for one month, and had as a consequence six months of disability. At the end of that time the injured sternoclavicular joint was still subluxated on lifting efforts and was a source of minor distress. The other patient was treated with adhesive pads across the joint surface and a sling for the forearm. A cross was applied, but the patient refused to wear it, and thirty-nine days after the injury, the deformity still recurred on raising the arm.

ACROMIO-CLAVICULAR DISLOCATIONS, COMPLETE

AGE YEARS	SEX	TREATMENT		DAYS OF DISABILITY	PERMANENT DISABILITY
		SLING, STRAPPING OR BANDAGE	BRACHIO-CLAVICULAR SPLINT		
30	Male	4 days	34 days	51 days	None-returned to work
29	Male	5 days none	30 days	49 days	None-returned to work
48	Female	———	35 days	56 days	None-returned to work
33	Male	Bed 15 days sling 15 days	Splint-55 days	120 days	None-returned to work
24	Male	———	Splint-35 days	46 days	None-returned to work
69	Male	———	Splint-42 days	84 days	None-returned to work
48	Male	———	Splint-22 days	75 days	None-returned to work
32	Male	Strapping 3 days	Splint-30 days	42 days	None-returned to work
57 *	Male	———	Splint-36 days	130 days	10 degrees loss forward flexion 20 degrees loss of abduction
17 (*)	Male	Strapping 26 days	———	47 days	Abnormal mobility still present-pain in heavy lifting.

Average Age-38.7 years.
Average Disability — 70 days.
Permanent Disability
* Head injury, marked scapulo-humeral, acromio-clavicular and spinal arthritis.
(*) 1932—Inadequate treatment by strapping only.

Chart III.

FRACTURE OF CLAVICLE INVOLVING ACROMIO-CLAVICULAR JOINT.

AGE YEARS	SEX	TREATMENT		DAYS OF DISABILITY	PERMANENT DISABILITY
		SLING, BANDAGE STRAPPING	BRACHIO-CLAVICULAR SPLINT		
29	Male	Sling 14 days	Splint 18 days	Alcoholic transient. Uncooperative Did not return.	Ulnar Palsy
34	Male	Sling 21 days	Splint 21 days	56 days	None-good function
47	Male	Velpeau bandage 6 days	Splint 5 weeks	180 days	(Head injury. Brachial Plexus neuritis, recovered.) None
67	Female	Velpeau bandage 4 days Inadequate splint for 2 weeks	Adequate splint 4 weeks	Still under treatment	
23	Male	Strapping and sling 4 weeks	No displacement of fragments. Fracture between joint and ligaments.	30 days	None-returned to work

STERNO-CLAVICULAR DISLOCATIONS

26	Male	Clavicular cross 1 month		49 days	None
33	Male	Aeroplane splint 6 weeks (referred from country) Sling 1 month.		6 months	Unstable joint Pain on lifting.
64	Female	(fracture) Clavicular cross 30 days		57 days	None
65	Male	(fracture) Adhesive strapping 23 days	Sling 14 days	37 days	—sent to Prison Deformity and subluxation still present. None.
50	Male	(fracture) Clavicular cross 4 weeks		8 weeks	None

Chart IV.

SUMMARY AND CONCLUSIONS

1. Acromioclavicular dislocations, complete or incomplete, can be adequately treated by simple means, and operation is unnecessary in most cases.

2. The retention apparatus must be worn for a time sufficient to allow the torn trapezoid and conoid, as well as the acromioclavicular ligaments, to heal in a shortened position and with normal adult tensile strength. Four weeks usually suffice.

3. A satisfactory simple method of maintaining reduction of the acromio clavicular joint dislocation is described.

4. The same means may be used in certain fractures of the outer end of the clavicle, in which tear of ligaments and deformity similar to that in tears of the acromioclavicular ligaments occurs.

5. Sternoclavicular dislocations while rare, can be maintained in reduction by the clavicular cross and normal joint movement and stability regained.*

REFERENCES

1. Jones, Sir Robert. Injuries to Joints, 2nd ed., p. 56. London, 1922. Henry Frowde and Hodder and Stoughton.
2. Mollier, S. Ueber die Statik und Mechanik des Menschlichen Schulter gurtels unter normalen und pathologischen Verhaltnisse. Festschrift zum Siebenzigsten Geburtstag von Carl von Kupffer. Jena, 1922. Gustav Fischer.
3. Bunnell, Sterling. Fascial graft for dislocation of the acromioclavicular joint. *Surg., Gynec. & Obst.*, 46: 563, 1928.
4. Henry, M. O. Acromioclavicular dislocations. *Minnesota Med.*, 12: 431 (July) 1929.
5. Bowers, R. F. Complete acromioclavicular separation. *J. Bone & Joint Surg.*, 17: 1005, 1935.

* Since this manuscript was submitted for publication, four complete acromioclavicular dislocations have been treated with the brachial clavicular traction splint. Three patients returned to work with no disability or deformity and with normal motion. The fourth, a patient who had a fractured carpal scaphoid of the same arm, finally came to operation.

ACUTE FRACTURES OF THE NECK OF THE FEMUR*

DEVELOPMENT OF THE TREATMENT

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TO general practitioners the recent changes which have occurred in the treatment of fractures of the neck of the femur must present a confusing picture. It is our object, therefore, to present a concise account of the evolution of the various methods of treatment and to indicate their general usefulness from a clinical point of view.

Among the first to draw attention to fractures of the neck of the femur was Ambroise Paré.[1] Although the injury was potentially coexistent with man himself, it had not been recognized as a clinical entity by earlier surgeons and even the great Paré looked on the condition as a traumatic dislocation. It was only after repeated unsuccessful attempts to reduce what he believed to be a dislocated hip that he arrived at the correct diagnosis of fracture of the femoral neck and instituted treatment by longitudinal extension.

Two landmarks divide the treatment of fractures of the neck of the femur into three eras. Each of the last two eras has been remarkable for the introduction of a new conception of the care of patients affected by this injury and each has been accompanied by an equally remarkable improvement in the end results of treatment.

Fractures of the neck of the femur may have been recognized by the ancient Egyptians, although no direct evidence of this has as yet been found. The first period of our arbitrarily divided phase of treatment therefore begins with the dawn of time and ends with the enunciation of the Whitman method of treatment in 1905.[31,32] The second period lasted from 1905 until 1931, and the third began with the general acceptance of internal fixation as the method of choice in the treatment of these fractures. This form of treatment still holds the field today among leading orthopedic surgeons.

The Edwin Smith[5] and Ebers[6] papyri are the earliest medical records, and it is to these that we must refer if we desire to have accurate information as to various methods of treating fractures in vogue during the first period. In the light of the means at their disposal the ancient Egyptians treated fractures excellently. Three forms of splinting were in everyday use: (1) Paired, bark splints padded with linen and moulded to the part. Retention of the splint was secured by bandages or linen ties. They were comfortable, secured rest of the part, and if their almost total absence from the Egyptian tombs is a criterion, gave excellent results. (2) The second variety might be considered the primordial ancestor of the present-day plaster cast. It was formed by the impregnation of several layers of linen by glue or resins and, while still soft, was moulded to the limb. It hardened to form an accurate mould of the affected part. A thousand years later we find that Hippocrates still used the same materials. (3) A third type of splint was a simple wooden peg which was used to gag the mouth open during tetanic convulsions so that feeding might be carried out.

In the early centuries the Arabians used a mixture of lime and egg white for making casts but, by 1798, they were using plaster of Paris. Hubenthal introduced plaster casts into Europe, but it was the Dutch

* Read in abstract before the meeting of the Watertown District Medical Society, Watertown, South Dakota, November 23, 1937.

army Surgeon, Mathijsen, who devised the plaster bandage in 1852. In France, splints of rushes were in use in the fifteenth and sixteenth centuries; in Japan and China, bamboo was requisitioned for a similar purpose because of its lightness, strength and durability. In times of war, necessity led to the use of a musket as a splint or crutch. From this it was but a step to the long Liston splint which provided not only for immobilization, but at the same time conserved the length of the limb. This splint consisted of a long wooden board, of sufficient length and breadth, which extended from the lower ribs to 4 inches beyond the foot. The first step in the application was to bind the leg to the splint and then, with the last turn or two of the bandage round the forked extremity of the splint, to secure the foot in good position. By means of a binder or bandage the splint was bound to the body and to the upper part of the thigh. Extension was obtained by means of a perineal band, which passed through two holes in the upper end of the splint in such a manner that, by tightening the band over the fixed perineum, the splint and leg could be pushed downward and so produce reduction. (Fig. 1.)

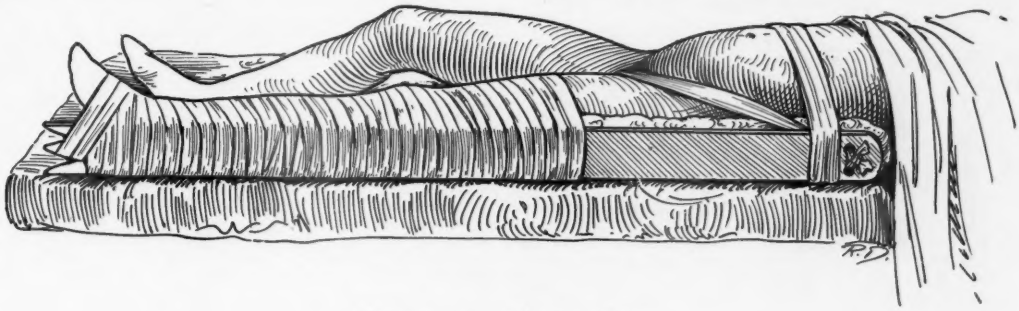

Fig. 1. Long Liston splint.

In 1850, Hugh Owen Thomas devised his hip and knee splints, a boon to all later surgeons. Seventy-seven years later, Meurice Sinclair stated that this (the Thomas splint and its modifications) had been directly responsible for the design of the majority of splints for the arm and leg which were used successfully in the World War. He added that it was "by far the finest piece of apparatus in our fracture equipment,"—a statement with which the present authors agree.

In spite of the appliances designed and the methods advocated in treatment, however, the general outlook regarding fractures of the hip in the latter part of the nineteenth century was gloomy in the extreme. As an index of the attitude of even the very elect in the profession toward fractures of the neck of the femur we can do no better than quote Sir Astley Cooper's own words: "Baffled in our various attempts at curing these cases, and finding the life of the patient occasionally sacrificed under the trials made to unite them, I should, if I sustained this accident in my own person, direct that a pillow should be placed under the limb throughout its length and that another should be rolled up under the knee, and that the limb should be thus extended until the inflammation and pain be subdued. I should then daily rise and sit in a high chair in order to prevent a degree of flexion which would be painful, and, walking with crutches, bear gently on the foot at first, then gradually more and more until the ligaments became thickened and the muscles increased in their power. A high-heeled boot should be next employed by which the halt would be much diminished." Such a statement from a man of Sir Astley Cooper's high professional standing meant that the most one could hope for was union by fibrous tissue, but at least one could avoid untimely death. In those days, and up until twenty-five years ago, every surgeon's purpose with respect to the treatment of fractures

was: (1) save the life of the patient, (2) obtain union, and (3) obtain good union. This was nowhere truer than in the case of fractures of the neck of the femur.

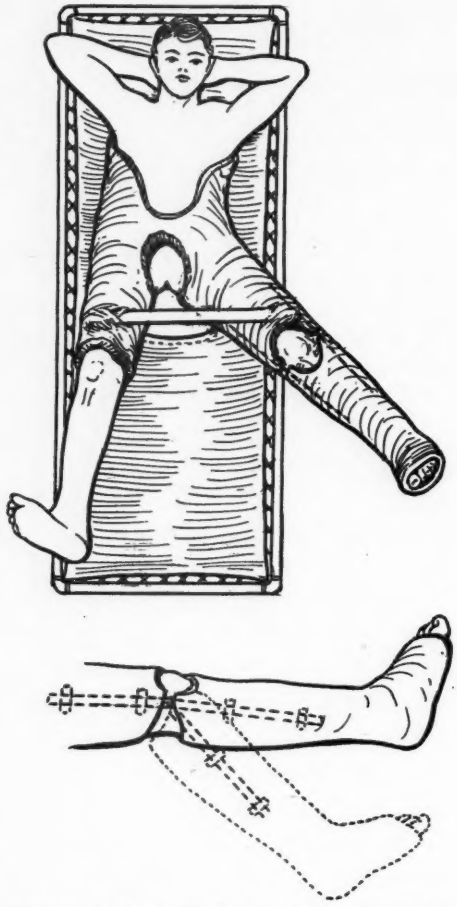

FIG. 2. Modified Whitman knee cast with hinge (Meyerding) at the knee joint.

Agnew, in 1878, in attempting to strike an optimistic note, succeeded only in intensifying the pessimistic attitude which was prevalent at the time. Speaking of the results of treating intracapsular fractures by the Astley Cooper method, he said: "There have been recorded a sufficient number of cases of bony union, after what was believed to be intracapsular fracture, to justify a hope that some of the cases encountered by the surgeon may have a similar termination." The doubt which surrounded the possibility of the healing of intracapsular fracture was widespread throughout Europe and claims of union were accepted *cum granum salis*.

Jonathan Hutchinson (1828–1913) in his description of the specimen of intracapsular fracture in the Pathological Museum of Leeds Hospital, wrote: "This specimen is alluded to by Malgaigne and Hamilton as if it were of doubtful validity, but neither of them had probably seen it. I cannot but hope that the publication of these life-sized drawings of the bone will set at rest all scepticism as to the possible union of intracapsular fractures. I trust, also, that it may lead to greater hopefulness in the treatment of those accidents and thus more systematic care in securing coaptation."

Indeed, so bad had the results of treatment been that men of the highest professional attainments had gradually come to regard all forms of active treatment as useless and in consequence had abandoned even the accepted methods of the day. This attitude of the profession prevailed to a great extent until 1905 when Royal Whitman introduced his method of fixation in plaster in wide abduction and internal rotation: "The patient, having been anesthetized, is placed on a pelvic support with a perineal bar, the shoulders resting on a box of equal height. Two assistants make manual traction on the extended limbs, drawing the perineum firmly against the bar and completely reducing the shortening on the injured side, the surgeon meanwhile lifting the thigh upwards if it is below the place of its fellow. The limb is then rotated slightly inwards, thus completely apposing the fragments. Both limbs, extended and under manual traction, are then abducted to the full limit, on the sound side first, to demonstrate the normal range, which varies in different individuals, and to balance the pelvis. When this limit is approached on the injured side, tension on the capsule assures the alignment of the fragments and forces a resistant contact. A long plaster spica extending from the axilla to the toes is then applied, which by fixing the limb in complete abduction, full extension, and slight inward rotation, assures the security

of the internal splinting. The knee is slightly flexed and the foot at a right angle with the leg slightly adducted."

Leadbetter modified this technique by reducing the fracture by perpendicular traction with the thigh flexed to a right angle; abduction and internal rotation followed. One important point in his method was his test for reduction. Reduction was complete when the affected foot remained upright on the palm of the examiner's hand. If external rotation occurred, reduction was incomplete and the maneuver had to be repeated. More recently (1936), Kleinberg, a former associate of Whitman, while adopting the Leadbetter technique, has modified the subsequent procedure by allowing patients, and one might almost say "forcing" patients, to get up on the third or fourth day and make attempts at walking. He claimed that by bearing weight on the affected leg, local union was not only encouraged but hastened.

There is no gainsaying the fact that union occurs in the cases of some old and frail individuals, but we believe that the last word has not been said on the subject of nonunion, its causes and its cure. We are convinced of the fact that old age per se is not the cause of nonunion any more than the fact that a patient has passed his allotted span makes him of necessity a poor surgical risk. We have treated patients in the seventh and eighth decades of life who have recovered from the effects of trauma which even in the robust and vigorous could have been adequate to produce death from shock. While the method employed in obtaining reduction of the fragments varies with the individual surgeon, means must be taken which will permit of reduction being maintained and at the same time prevent the development of pressure sores and hypostatic pneumonia. By the use of the plaster cast this is possible. The patient may be moved into any position or even transported in comfort. The stiffness of the knee so commonly met with in these elderly, often arthritic, patients following prolonged fixation in the Whitman cast can be prevented by employing a certain type of knee hinge in the cast as described by Meyerding. (Fig. 2.)

A commission appointed by the American Orthopedic Association to study the end results of the Whitman treatment of intracapsular fractures of the neck of the femur reported in October, 1930 that 50 per cent of patients under the age of 60 could look forward to solid bony union, but that the percentage of union in those more than 60 years of age was distinctly lower. While all credit must go to Royal Whitman for his excellent practice and advocacy of the abduction and fixation treatment, we must concede a modicum of praise to Hennequin and to Newton Schafer who were probably the first in the field to use the method. In 1927, Cotton advised artificial impaction by means of a mallet following reduction as a means whereby union could be made more certain.

In 1850, Lagenbeck made the first attempt to treat fractures surgically by internal fixation. He used a silver drill to unite the fragments, but old age and "hospital gangrene" unfortunately defeated his ingenuity and surgical heroism. Lister, some years later, made a similar attempt. The details given of the operation were insufficient and we are not certain that the fragments were pegged. Patients survived but union by fibrous tissue alone was obtained. If the fragments were not pegged, this was probably the first occasion on which drilling of the head and neck of the femur was carried out.

The first report of a successful case was published by König in 1878. The patient was a young and vigorous man who was operated on under aseptic conditions through a small lateral incision over the greater trochanter. Between 1878 and the adoption of the more modern forms of internal fixation much work was done in this field. Murphy and Beckman in this country and MacLennan in Scotland used ordinary nails and screws, with variable success. Bone pegs of the intramedullary

FIG. 3. Showing Smith-Peterson nail in place.

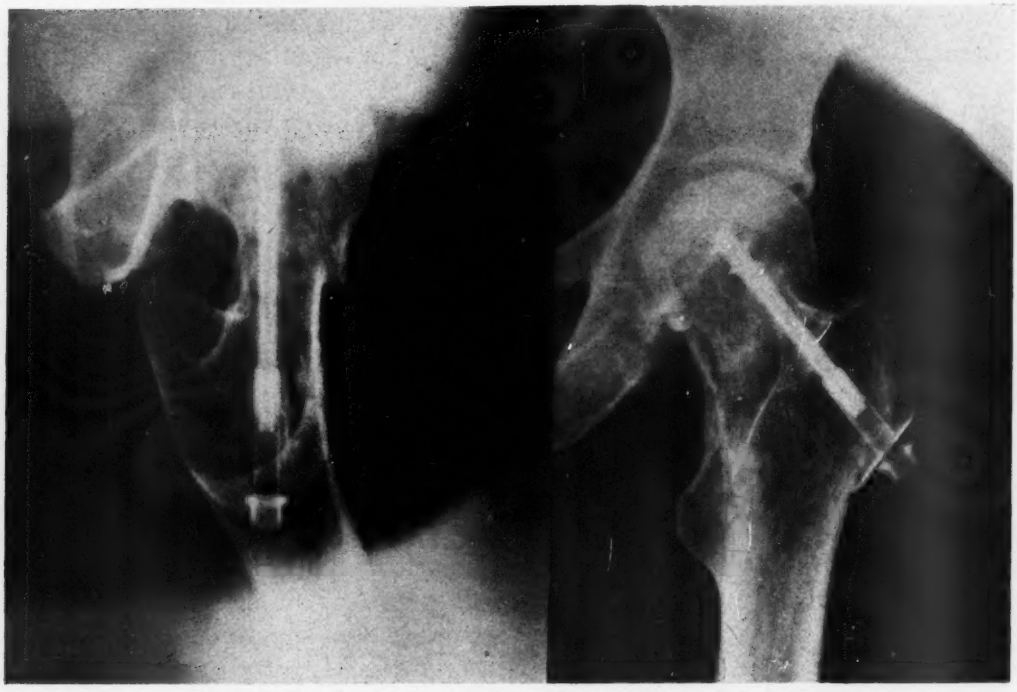

FIG. 4. Showing the lag screw (as described by Henderson).

variety, or bone grafting operations in which the tibia and fibula were used, have been employed and advocated by Albee, Henderson,[13] and Campbell although these procedures have a greater sphere of usefulness in the treatment of ununited old fractures of the femoral neck. The nail used originally by Smith-Petersen was a solid one and the difficulty of introducing it was considerable, necessitating arthrotomy if accurate placing in the head of the femur was to be secured. With the modification suggested independently by Johansson in Sweden, King in Melbourne, and Henderson[12] at The Mayo Clinic, whereby a cannulated nail was used, the difficulty of introduction and the severity of the operation were immensely reduced.

In all our surgical treatment the fracture was reduced by simple Buck's (1861) extension or by either the Whitman or Leadbetter maneuvers. The accuracy of the reduction was checked by anteroposterior and lateral roentgenograms. The modern use of roentgenograms permits of accurate determination of the position of the fragments following attempts at reduction and before internal fixation is employed. This, performed under spinal anesthesia, and by a surgical team experienced in the technique, results in a lessened mortality rate. A Kirschner wire was then introduced, through a small lateral incision centered over the lower border of the greater trochanter, by drilling up through the neck into the head. Localization of the wire could be accurately determined by films or fluoroscopic examination. The Smith-Petersen nail was then slipped over the wire and driven home through the femoral neck and into the head. The immediate and late effects, while excellent, were not perfect, but the percentage of good end results was definitely increased for all ages and a new day had dawned for the unhappy sufferer from this type of fracture. (Fig. 3.)

There are two disadvantages, however, to the use of the Smith-Petersen nail: (1) in some cases it slips out; (2) in inserting the nail, separation of the fragments may occur so that impaction subsequently is necessary. To overcome these defects Henderson[11,12] suggested the use of the lag screw. This screw has been in use among furniture makers for many years and is mechanically adapted to overcome just these disadvantages. The method of introduction is similar in all respects, but there are a few points upon which Henderson lays special emphasis: No attempt at manipulative reduction has been necessary since reduction has been accomplished in all cases by Buck's extension.

We believe in waiting until a week has elapsed following injury before surgical intervention is undertaken, even though no displacement has occurred. The advantages gained from this procedure outweigh anything that may be lost by waiting. Shock has been combated, the vital forces of the aged and frail have been fortified, and the possibility of an undiscovered yet potentially great surgical risk eliminated. Deaths which occur during this period of waiting can, therefore, be accurately classified as being due to primary shock or debility and not used as reasons for discrediting an otherwise life-saving surgical procedure.

With accurate coaptation of the fractured ends, confirmed roentgenologically, and with the lower border of the greater trochanter exposed through a small lateral incision, a Kirschner wire is run into the femoral neck and through the head until it impinges on the acetabular wall. No trouble results from this minor trauma to the articular cartilage and a firmly fixed proximal fragment is obtained which renders the subsequent steps partially to drill the neck and head simple and accurate. The insertion of the lag screw along the Kirschner wire is straightforward. When it is well buried in the head, the covering sleeve is introduced by special instruments and tightened down upon the screw and, as this occurs, the fragments are drawn together so that firm and accurate coaptation is obtained. No retention plaster cast

is required—a simple foot splint with a cross bar being adequate to prevent external rotation of the leg. (Fig. 4.)

Within two weeks flexion movements at the hip by means of a knee sling are begun but, later on, are carried out actively. The patient is allowed up in six weeks on crutches, although weight-bearing is not attempted until after the third month. Ordinarily we believe that removal of the screw in about four months prevents excessive absorption of the neck of the femur and, by removing a foreign body, militates against subsequent trouble and electrolytic reaction. In the series of cases at The Mayo Clinic in which treatment was conducted by this method, bony union occurred with excellent functional results in about 72 per cent.

Today, each orthopedic surgeon follows the method which appears to him to be the most effective, and whether it be that entailing the use of a Smith-Petersen nail, a Henderson lag screw, Moore's pins, or Martin's screws, in the hands of a capable man the end results are considerably better than those which followed the older forms of simple fixation. It is no simple matter to insert a nail with certainty, and many have been the devices tried to render this primary operative step one of mathematical accuracy. Valls of Buenos Aires and Hey-Groves of Bristol have devised ingenious inserters which make the operative technique simpler.

SUMMARY

The subject of fracture of the neck of the femur has been reviewed from its recognition at the time of Ambroise Paré, through the period of the epoch making changes introduced by Whitman, up to the modern method of internal fixation.

In cases of fresh fracture of the neck of the femur the use of the Smith-Petersen nail and of the Henderson lag screw, under the control of Roentgen rays, has resulted in accurate reposition of the fragments and their complete fixation, with subsequent bony union in 72 per cent of cases. The excellent results which have been obtained by modern treatment of this most disabling and crippling condition have changed the pessimistic attitude, with which the profession as a whole regarded this injury in the late nineteenth century to the one of present day optimism.

REFERENCES

1. Agnew, D. H. Injuries and diseases of the osseous system. Intracapsular fractures of the femur. In: Principles and Practice of Surgery, vol. 1, p. 938. Philadelphia, 1878. J. B. Lippincott.
2. Albee, F. H. Injuries and Diseases of the Hip. New York, 1937. Paul B. Hoeber.
3. Beckman, E. H. Personal communication.
4. Bick, E. M. Quoted by Paré, A. In: The Source Book of Orthopaedics. Baltimore, 1937. Williams and Wilkins.
5. Breasted, J. H. The Edwin Smith Surgical Papyrus, vol. 1. Chicago, 1930. University of Chicago Press.
6. Bryan, C. P. The Papyrus Ebers. London, 1930. Garden City Press.
7. Buck, Gordon. Description of an improved extension apparatus for the treatment of fracture of the thigh, in use in the New York Hospital for the past six years, and in the United States army general hospitals during the Civil War. *M. Rec.*, 2: 49–51 (March 15) 1867.

7a. Campbell, W. C. Fractures of the neck of the femur. *New Internat. Clin.*, 1: 34–43 (March) 1938.

8. Cooper, Astley. Quoted by Senn, N. Surgical Monographs. Fractures of the Neck of the Femur. Philadelphia, 1883. Collins.
9. Cotton, F. J. Artificial impaction in hip fractures. *Surg., Gynec., & Obst.*, 45: 307–319 (Sept.) 1927.
10. Groves, E. W. H. Fractures of the neck of the femur. *Brit. M. J.*, 2: 491–493 (Sept. 14) 1935.
11. Henderson, M. S. Internal fixation of fractures of the neck of the femur. *Arch. Surg.*, 35: 419–430 (Sept.) 1937.
12. Henderson, M. S. Internal fixation for recent fractures of the neck of the femur. *Ann. Surg.*, 107: 132–142 (Jan.) 1938.
13. Henderson, M. S. Bone grafts in ununited fractures. *J. Bone & Joint Surg.*, 20: 635–647 (July) 1938.
14. Hubenthal. Quoted by Bick.[4]
15. Hutchinson, J. Quoted by Senn, N. Surgical Monographs. Fractures of the Neck of the Femur. Philadelphia, 1883. Collins.
16. Johansson, Sven. On the operative treatment of medial fractures of the neck of the femur. *Acta orthop. Scandinav.*, 3: 362–392, 1932.
17. Kleinberg, Samuel. The value of early weight-bearing in the treatment of fractures of the neck of the femur: with a report of twenty-four cases. *J. Bone & Joint Surg.*, 19: 964–977 (Oct.) 1937.
18. König. Report before seventh Congress. *Verhandl. d. deutsch. Gesellsch. f. Chir.*, 1878, p. 93.

19. Langenbeck. Quoted by Senn, N. Surgical Monographs. Fractures of the Neck of the Femur. Philadelphia, 1883. Collins.
20. Leadbetter, G. W. Non-operative treatment for fracture of the neck of the femur. *Am. J. Surg.*, 38: 612–618 (Dec.) 1937.
21. Lister. Quoted by Senn, N. Surgical Monographs. Fractures of the Neck of the Femur. Philadelphia, 1883. Collins.
22. MacLennan, A. Personal communication.
23. Mathijsen. Quoted by Bick.[4]
24. Meyerding, H. W. The non-operative treatment of recent fractures of the femur. *Minnesota Med.*, 7: 237–247 (April) 1924.
25. Murphy, J. B. Personal communication.
26. Report of a commission appointed by the American Orthopaedic Association to study the end results of intracapsular fractures of the neck of the femur. *J. Bone & Joint Surg.*, 12: 966–969 (Oct.) 1930.
27. Sinclair, Meurice. The Thomas Splint. London, 1927. Oxford University Press.
28. Smith-Petersen, M. N., Cave, E. F. and Vangorder, G. W. Intracapsular fractures of the neck of the femur; treatment by internal fixation. *Arch. Surg.*, 23: 715–759 (Nov.) 1931.
29. Thomas, H. O. Fractures, Dislocations, Deformities and Diseases of the Lower Extremities. London, 1890. H. K. Lewis.
30. Valls, José, and Lagomarsino, Enrique. Technique personnelle et nouvelle instrumentation pour l'ostéosynthèse du col fémoral à ciel ouvert. *Bull. et mém. Soc. nat. d. chir.*, 61: 1204–1208, 1935.
31. Whitman, Royal. A treatment of fracture of the neck of the femur designed to improve functional results by immediate reduction of deformity and by effective protection during the period of repair. *Am. J. M. Sc.*, 130: 1–21 (July) 1905.
32. Whitman, Royal. The abduction treatment of fracture of the neck of the femur. In: Orthopaedic Surgery. Philadelphia, 1923, Lea & Febiger.

We understand physical pain aright only when we realise the part played by the individual in a system morphologically the same for everyone, when we have analysed . . . *living* pain.
From—"Surgery of Pain" by René Leriche (Williams & Wilkins).

TREATMENT OF DELAYED OR NON-UNION BY BONE DRILLING

STANLEY M. LEYDIG, M.D.

ST. LOUIS, MISSOURI

DURING 1937 330 fractures of the tibia (excluding Pott's fractures involving the medial malleolus)

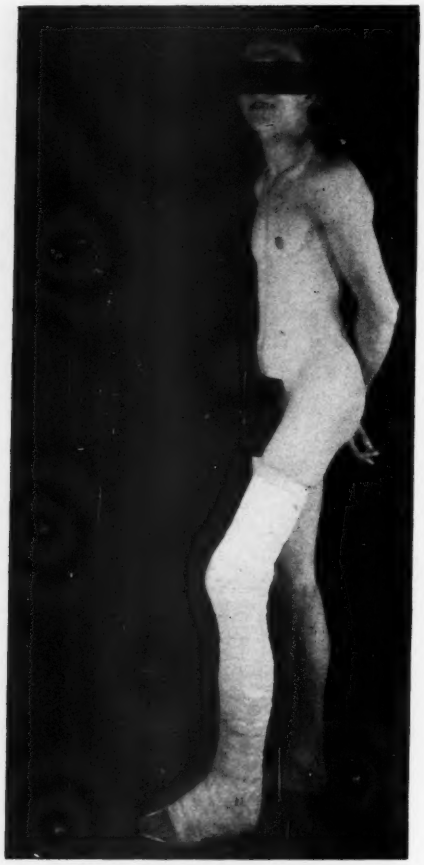

FIG. 1. Case III. Mid-thigh non-padded cast with iron walking caliper, applied after bone drilling.

were treated at St. Louis City Hospital. Of this number eight, or 2.4 per cent, were considered to be cases of delayed union or potential nonunion. No apparent mechanical factors were involved, such as interposition of fascia or muscle between the fragments, nor was there any derangement of the calcium metabolism as far as could be determined by blood chemistry.

The probable causes of the delayed union in these cases were: (1) poor approximation of the fragments at initial reduction; (2) remanipulation due to slipping of the fragments; and (3) the fact that some were compound.

In our experience a compound fracture usually is slower to unite than a simple one. One probable explanation is the loss of blood which is a simple fracture, remains as a hematoma around the fracture site and possibly serves as a medium for invading osteoid tissue and aids in the liberation of phosphatase (which is considered essential in the deposition of calcium[1]). Contrary to popular opinion, the age of the patient has no relation to nonunion; in our eight cases the age varied from 20 to 69.

In our experience the repair of fractures of the shaft of the tibia usually requires twelve to fourteen weeks of immobilization, and some cases require an even longer time. Usually, however, weight-bearing in a non-padded "skin" cast with an iron walking caliper has promoted union when it did not appear likely to occur after three months. This régime has been successful in all but eight cases, in which the fragments were drilled. Previously, such cases underwent surgical intervention in which either bone grafting, resection of the fibrous union, or plating was done. In the past year, since we resorted to bone drilling, not a single bone graft or similar procedure has been necessary for nonunion of the tibia.

The treatment of nonunion by bone drilling was first proposed by A. Beck[2] of Kiel, and described by Lorenz Böhler[3] of Vienna. Apparently this procedure is not generally recognized in this country, as few references commending it are found in the literature. This principle has been used, however, for treatment in fractures of the neck of the femur and Legg-Perthe's disease,[4] ununited fractures of carpal bones,[5] and recently a series of cases of the long bones.[6]

The indications for drilling are: persistent mobility after adequate immobilization, as detected clinically; lack of new bone formation bridging the fracture site, as determined by *x*-ray. The contraindications are the same as for any bone surgical procedure. We prefer to confine the drilling to the bones of the leg or lower thigh, because there is less possibility of injuring other important structures than in the arm. We do not wait so long as we formerly did before resorting to drilling the fragments, as it is known that prolonged maintenance of the fragments in poor alignment prevents an early return to normal function.

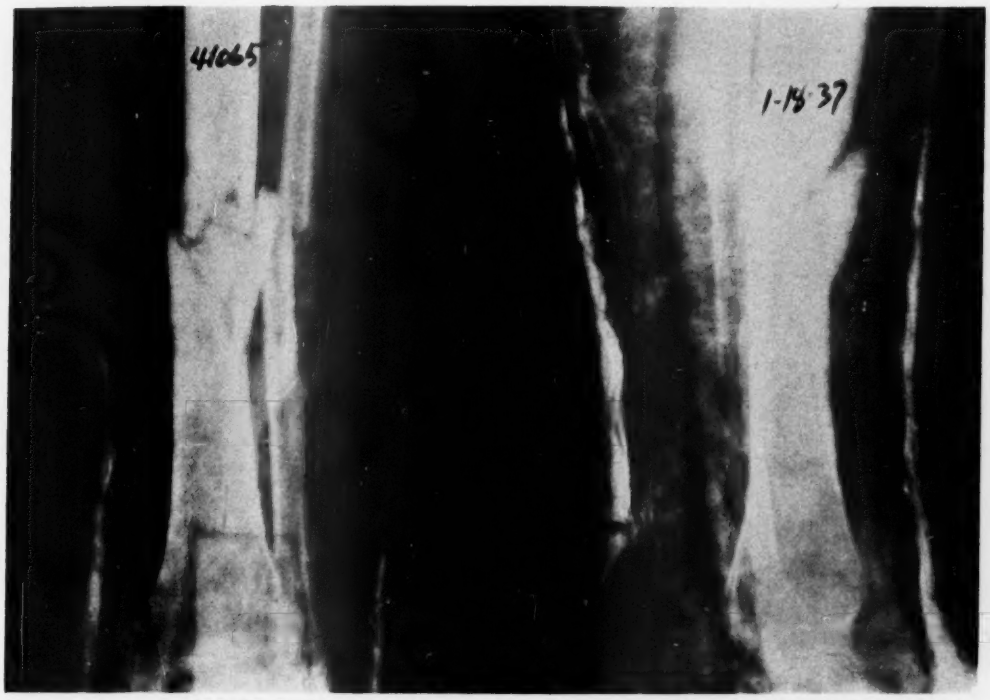

Fig. 2. Case III. Mid-third of tibia with nonunion after twenty-nine weeks immobilization in cast.

Bone drilling is done in the fluoroscopy room. However, although the fluoroscope is helpful, it is not necessary. The skin over the nonunion is prepared with iodine and alcohol, and infiltrated with novocaine (½ per cent). The periosteum must also be

TABLE I

Site of Fracture	Age	Time of Immobilization, Weeks	Possible Cause of Nonunion	Time of Immobilization after Drilling before Union Occurred, Weeks
Junction mid and distal third of tibia	69	24	Compound	4
Mid third of tibia	23	18	Undetermined	4
Mid third of tibia	20	29	Undetermined	4
Junction mid and distal thirds of tibia	21	17	Poor reduction	5
Junction mid and distal third of tibia	48	28	Compound	5
Junction mid and distal third of tibia	40	42	Remanipulation three times	5
Distal third of femur	62	63	Poor approximation and compound	6
Mid third of tibia	69	17	Remanipulation twice	9
Mid third of tibia	54	12	Poor approximation	8

injected carefully or the patient will complain. Two or three puncture wounds are made over the proximal and distal fragments adjacent to the fracture site, about ½ inch apart, through which the drill is inserted. Eight to ten holes are then drilled obliquely into the bone with a 3/16 inch drill, in such a way as to traverse both fragments with each drilling. This is easily done in spiral or oblique fractures, but in transverse fractures more care is required. A non-padded ambulatory plaster cast with an iron walking caliper is applied over a sterile dressing. The patient is encouraged to walk on the cast the next day, after the plaster is dry and the slight pain has subsided.

Table 1 shows the results of eight cases of nonunion of the tibia and one of the distal third of the femur.

FIG. 3. End result of same case after drilling. New bone had formed, and fracture was clinically solid in four weeks. Patient allowed full weight bearing after two months.

CONCLUSIONS

Delayed or nonunion is not uncommon in fractures of the shaft of the tibia, and seems to follow more frequently the fractures which were (1) not accurately reduced, (2) re-manipulated, or (3) compounded.

Bone drilling in such cases offers a simple method of promoting union, and has supplanted major surgical procedures for delayed or nonunion of the tibia in the St. Louis City Hospital.

Eight patients so treated required solid union clinically, and the *x*-ray revealed new bone formation within a few weeks.

REFERENCES

1. MURRAY, C. R. Delayed and non-union in fractures in adult. *Ann. Surg.*, 93: 961, 1931.
2. BECK, A. Zur Bedhandlung der verzogerten Konsolidation bei Unterschenkelbruchen. *Zentralbl. f. Chir.*, 56: 2690, 1929.
3. BÖHLER, L. The Treatment of Fractures. Baltimore, 1935. William Wood.
4. BOZSAN, E. H. A new treatment of intracapsular fractures of neck of the femur and Calve-Legg-Perthe's disease. *J. Bone & Joint Surg.*, 14: 884, 1932.
5. SPEED, K. Small bone repair. *Surg., Gynec. & Obst.*, 64: 9, 1937.
6. EASTON, E. R., and PREWITT, P. V. Treatment of ununited fractures by bone drilling. *J. Bone & Joint Surg.*, 19: 230–231, 1937.

THE USE OF REGIONAL NERVE BLOCK DURING TREATMENT FOR FRACTURED RIBS*

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FRACTURE of the ribs, in the large majority of cases, is within easy management. Uncomplicated fractures may be treated by simple adhesive strapping for immobilization of the rib fragments. Such therapy usually terminates favorably within three to four weeks. The most annoying complication during such treatment is pain. Adhesive strapping may serve well to maintain proper alignment of the broken bone fragments and to diminish respiratory movements of the chest, but it will not entirely eliminate motion. With motion some degree of pain will regularly be present at the site of fracture.

Pain may be only an annoying disturbance during healing of rib fractures, but it can be the primary cause of more serious sequelae. The more serious complications following simple fracture of the ribs are respiratory infections. Most of the few patients with simple rib fracture terminating fatally, die from pneumonia. The incidence is fairly high in the aged. Maingot has often observed pneumonia following the fracture of one or two ribs in elderly patients.[1] Ten Horn also found pneumonia responsible for most deaths after fracture of the ribs. In elderly individuals, he recommends that no attempt be made to immobilize the chest mechanically. His main reliance is on morphine given in large doses and repeated often enough to control the pain. He insists, "that morphine in addition to relieving the pain, permits the respirations to proceed unhampered and reduces the need for oxygen, but its greatest benefit is the prophylaxis against pneumonia by allowing coughing without pain."[2] Pain may continue for a considerable time after the fracture of a rib. Persistent pain is most often callus pain.[3] Rarely is it a generalized intercostal neuralgia although the latter is sometimes reported. Regardless of the cause, as long as pain is present at or near the site of fractured ribs the patient is more likely to develop pneumonia than would be true were the pain relieved.

Extensive fractures, those involving several ribs, and those complicated by much overlapping of fragments, traumatic pleurisy, hemorrhage, tension pneumothorax, emphysema of the skin, etc., present therapeutic indications entirely different from simple fractures. It also follows that in this group an unfavorable issue is not uncommon. In severe crushing injuries of the chest, even those without severe hemorrhage or torn lung, the mortality is very high.[4] Fatalities, when they occur several days after the injury, are often due to pneumonia. In patients with extensive or complicated rib fracture severe pain is regularly a persistent complication. In this group pain becomes a complication of major importance and it has a definite bearing upon the mortality. Because of the severe pain these patients put forth every effort to eliminate any activity that will increase their discomfort. Coughing is suppressed, movements that involve the trunk are not undertaken, and deep breathing is avoided. Respiratory effort may be so

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retarded that the involved chest is practically splinted. It has been observed that actual massive collapse of the lung may follow such forced inactivity. It regularly happens that the application of plaster bandage or even a cast fails to alleviate the pain and sedatives or hypnotics must be administered.

Since pain may play such an important rôle in mortality following severe trauma to the chest, it seems logical to attempt the relief of pain early in the treatment. It has been pointed out that the control of pain or adequate sedation, in an amount that will not seriously depress respirations, is necessary in chest wounds requiring operative intervention.[5] Generous amounts of morphine or other opiates are frequently given. Their use is not advised although their favorable effect upon pain is fully realized. The relief of pain in traumatic chests has for its therapeutic usefulness not only alleviation of the patient's discomfort but also the more important objective, prophylaxis against serious or fatal pulmonary complications. To serve this purpose it is necessary that the patient increase the ventilation of his lungs by deep breathing and that the lower respiratory tract be kept free from secretions by coughing. The opiates do not favor increased ventilation or vigorous coughing. The depression of respiration that follows the large amounts of these drugs required adequately to relieve the severe pain accompanying chest injuries, decreases ventilation of the lungs and seriously impairs the cough reflex. A more logical method for pain relief is the interruption of painful stimuli along their nervous pathways. The use of regional nerve block provides a means of adequate therapy for pain. It is without the objections raised against the opiates. It is obvious of course, that such a therapeutic measure as nerve block is no substitute for surgical therapy in cases with severe pleuropulmonary injuries but should be employed only as an adjunct.

Regional anesthesia designed to control pain should be directed toward paralyzing the nerves supplying the site of painful stimuli. With pain arising from a fractured rib, the intercostal nerve corresponding to the involved rib would immediately be suggested for injection. The injection of a single intercostal nerve does not invariably produce complete anesthesia along the entire course of the corresponding rib. The depression of painful stimuli may be so profound that complete relief is obtained or it may follow that accessory filaments from contiguous nerves will prevent more than a lessened pain response. To insure insensibility over the entire extent of a fractured rib it may frequently be necessary to inject not only the nerve corresponding to the fractured bone but also the nerves above and below the injured rib when intercostal block is employed. The use of paravertebral nerve block will more nearly insure complete pain relief.

The technique for paravertebral injection of thoracic nerves is not complicated. Excellent descriptions of the recommended procedures are given by Labat.[6] The technique employed here is essentially that described by Labat as his technique No. 1. The patient is placed upon the uninjured side and the knees flexed so as to widen the intercostal spaces. The spinous processes of the dorsal vertebrae are defined. Opposite each spinous process corresponding to the intercostal space selected for injection, a skin wheal is raised with procaine solution. The distance of the wheal from the midline varies from 4 to 5 cm. depending upon the width of the patient's back. An 8 or 10 cm. small gauge needle unattached to the syringe is introduced through the wheal and advanced at right angles with the skin surface so as to contact the rib just above the intercostal space to be injected. The rib is contacted and the needle is slightly withdrawn so that its direction may be changed. The syringe end of the needle is inclined outward and upward until the angle formed between the skin surface and the shaft of the needle is approximately 45 degrees. It is then advanced about 1 cm. downward and

inward toward the lower border of the rib. The lower border of the rib is reached by the point of the needle with this maneuver and it is possible by slightly changing the direction downward to feel the needle slip off the rib. With the point of the needle located just below the rib it is advanced inward and downward across the intercostal space, a distance of approximately 2 cm., where it impinges upon the body of the vertebra. The syringe is then attached and injection of the anesthetic solution begun after withdrawing the needle a very short distance from the bony surface and carefully performing the aspiration test.

The dangers of this procedure have been described by some as those attending puncture of the pleural cavity, the lung, an intercostal blood vessel or the subarachnoid space. None of these accidents should result in serious complications and their occurrence is not common. Puncturing the pleural cavity with a small needle will produce no serious complication and injection of solutions will do little more than induce coughing. Pneumothorax is unlikely to follow unless the pleura is torn and an aperture larger than that produced by a needle puncture is made. It is advisable to inject very slowly at the start and if coughing is induced, stop the injection until the needle can be properly placed. If the needle is inadvertently inserted into lung tissue, blood will follow the aspiration test. Injecting a small amount of anesthetic solution into the lung causes little more than a bitter taste in the patient's mouth. If an intercostal blood vessel is punctured, blood will be aspirated from the needle. The small hematoma that will be created, after puncture with the fine needle used for nerve blocking, is without significance. Although it is practically impossible to introduce a needle through an intervertebral foramen when the described technique is followed, occasionally cerebrospinal fluid is aspirated after the needle is put in place. The explanation for such an occurrence is the puncture of an abnormal prolongation of the subarachnoid cul-de-sac accompanying the nerve beyond the intervertebral foramen. If the technique is carefully followed and the aspiration test in two planes completely carried out and frequently repeated the dangers accompanying paravertebral nerve block are remote.

One per cent solution of procaine hydrochloride is recommended for paravertebral nerve block. Five to 6 c.c. may be placed at each site of injection. Stronger solutions may be too toxic when a considerable amount is used to secure extensive anesthesia. Weaker solutions, to be effective, must be injected in volumes too large for convenience. Five or 6 c.c. of fluid diffuse in the intercostal space and comes into contact with both anterior and posterior primary divisions of the nerve as well as the communicating rami.

When alcohol is to be injected it should always be preceded by the procaine block as described, but with a smaller amount of solution. Three or 4 c.c. of 1 per cent procaine solution will be sufficient. The alcohol should be injected fifteen to twenty minutes later. The position of the patient and of the needle should not be changed during the interval between injections. The initial injection of procaine will produce an anesthetized area upon which to place the temporarily irritating alcohol and its rapid absorption will eliminate diluting the alcoholic solution so that it may not be of maximum effectiveness. With this technique 1 c.c. of 95 per cent or absolute alcohol gives satisfactory results. More than 2 c.c. of alcohol should not be needed if properly placed and this amount should never be exceeded.

Aside from those complications already discussed, that may follow during the technical procedure of thoracic paravertebral anesthesia and those resulting from errors in technique, no serious objections other than those relating to the toxicity of procaine have been advanced. When alcohol is employed pleural irritation from injury with the needle, together with an inflammatory reaction caused by the

alcohol, will infrequently follow the injection. The predominating symptom is a burning pain which is noticed at about the time anesthesia from the procaine is lost. A small minority of patients may develop irritation neuritis of the somatic nerves. Such a neuritis may occur at any time for a week after injection and varies greatly in intensity. Either complication may be satisfactorily treated by procaine infiltration of the nerves which are distributed to the thoracic segments involved.[7] Hydrothorax, due to pleural irritation from alcohol, has been reported as a rare complication.[8] The contention that intercostal nerves are permanently destroyed with alcohol is not supported clinically or experimentally. The paralysis is transitory and seldom exceeds two or three weeks. The fine sympathetic rami may not regain function.[9] The possibility of further tissue injury from broken bone fragments in painless areas may be obviated by strapping or the application of a light cast which should be a regular part of the treatment. It has been demonstrated by Latteri[10] that the use of alcoholic injections during treatment of fractured ribs in no way affects the formation and progress of the callus or the proper healing of the fracture.

Paravertebral nerve block has recently been employed here in the treatment of crushing injuries of the chest. Thirteen patients with severely complicated rib fractures have been treated. Without exception these patients were admitted to the hospital by ambulance and were considered serious emergencies. For each of them prognosis was reserved until the extent of the respiratory complications might be determined following several days' hospitalization. The fractures involved from one to ten ribs, and five of the patients had accompanying fractures of other bones. Pulmonary complications included consolidation, atelectasis, tension and hemopneumothorax, subcutaneous emphysema, hemorrhage and various other pleuropulmonary injuries. Cyanosis, dyspnea and orthopnea were usually present at the time of admission. Severe, continuous pain was a prominent symptom of every patient.

Paravertebral injections of anesthetic solutions was accomplished soon after a diagnosis of the extent of the injury was tentatively made. The early cases received procaine block. Patients with fracture of three ribs or less were injected with a 2 per cent solution. When more extensive block was required, 1 per cent solutions were employed. The therapeutic results of regional anesthesia were immediate and of definite usefulness. Pain was completely relieved for eight of the patients, suppressed to the extent that ordinary breathing was not uncomfortable for four more, and made less severe for one individual. Following the anesthetic nerve block each of the patients could be moved about without serious complaint and would carry out instructions to breath deeply and cough. The duration of pain relief from procaine injections varied from three to twelve hours. It was the practice, with the first use of this therapy, to repeat the nerve block with the return of pain. Later cases received alcohol following the technique already described. One patient had five treatments with procaine. No patient receiving alcohol required more than one nerve block.

Two of the thirteen treated patients died as a result of complications from their injury. One expired on the seventh day after admission. This patient had fractures of both femurs, and the left ulna, lacerations of the spleen and left kidney and extensive bronchopneumonia. Another died on the eighteenth day after accident. Autopsy revealed lacerations of the lung, pulmonary emboli and hemorrhage of the lesser omental sac.

The results that may follow nerve block procedures during treatment of fractured ribs are illustrated by the two following cases. These cases are selected as the two most favorable results in the brief series reported.

Case 1. A white male, age 58, entered the hospital from the ambulance with marked

emphysema and external evidence of injury to the left chest. There was profuse epistaxis. The accident had occurred the day before admission. The diagnosis was multiple rib fractures (third to ninth inclusive) on the left side, subcutaneous emphysema on the left from neck to scrotum, and consolidation of the right chest. The patient was in severe pain, very cyanotic, dyspneic and orthopneic. He would not cough or tolerate moving because of the pain. Oxygen therapy and a moderate amount of morphine did not improve the condition. A paravertebral procaine block was completed on the left side to include the third to eleventh thoracic nerves. Pain relief was immediate and complete. The patient coughed freely, was moved from side to side and the consolidation in the right chest could no longer be diagnosed with certainty. The cyanosis cleared and breathing assumed a normal character. Severe pain returned after sixteen hours. The procaine injection was repeated and again gave complete relief. After eight hours a third block was done. Thereafter pain was not a prominent symptom. *X*-ray examinations then revealed "irregular patches of pneumonitis in the right thorax." Recovery was complete in five weeks.

Case II. A white male, age 60, was admitted from the ambulance at noon. He had been injured at 4 A.M. He was cyanotic and dyspneic with subcutaneous emphysema from the clavicle to the lower chest on the left side. The left ribs (sixth to ninth inclusive) were fractured in the midaxillary line. There was evidence of hemopneumothorax of the left chest. Late on the day of admission the temperature was 102 degrees and cyanosis and dyspnea were more severe despite oxygen therapy. Examination revealed signs of "atelectasis or bronchopneumonia of the right lower lobe posteriorly, related to stasis of secretions." Pain was severe and deep breathing or coughing was suppressed. A left paravertebral nerve block was performed with procaine to include the fifth to the tenth thoracic nerves. The pain was immediately eliminated and cyanosis and dyspnea were relieved. The patient was turned to the right side and coughed vigorously without severe discomfort. He was comfortable throughout the night. The anesthesia was repeated on the following morning when less severe pain had returned. Another block was done in the late evening. There was no complaint of severe pain at the fracture site after the third block but forced coughing caused some discomfort. More than 300 c.c. of blood was removed from the left chest. Seven days after injury, examination revealed "a residual hemopneumothorax and resolving bronchopneumonia progressing favorably." Recovery was complete.

No reference has been found to the use of paravertebral nerve block for the relief of pain from fractured ribs. Intercostal nerve block has been employed for ambulatory patients during the treatment of uncomplicated rib fracture. Leotta has treated pain associated with tuberculosis by alcohol injection of the involved intercostal nerves.[11] His technique was applied successfully in four cases of rib fracture by Latteri.[10] Grieco selected a group of uncomplicated cases of rib fracture which ordinarily would be treated ambulatorily. Patients were chosen in whom pain was persistent despite immobilization by adhesive strapping. He concluded after treating ten patients that intercostal nerve block with alcohol afforded quick relief from pain and considerably shortened the period of disability.[12] The same therapeutic procedure was used by Rabboni to treat ten ambulatory patients.[13] Pain relief was obtained for each individual treated and nothing developed to complicate the initial lesion or its treatment. Zoppi has recommended the daily subcutaneous injection of procaine at the site of fractured ribs to allay pain and eliminate the use of morphine.[14] The experience here has not included the treatment of ambulatory patients with paravertebral block, but the favorable effect upon pain for nonambulatory patients, the simplicity of the technique and absence of complications definitely recommend it.

SUMMARY

The treatment of simple and complicated fractures of the ribs should include some therapeutic medium for relieving the associated pain. The relief of pain is important for the patient's comfort. It is more important as prophylaxis against respiratory infections. Respiratory infections are re-

sponsible for the majority of deaths that follow these injuries.

The use of large amounts of opiates or narcotics to treat the pain associated with fractured ribs is not advised since adequate lung ventilation may be hindered and the stasis of secretions enhanced by depressing respirations and the cough reflex.

The use of paravertebral injections of procaine and alcohol at the site of the involved thoracic nerves is recommended. A technic for the convenient application of the regional nerve block is described.

Results following the treatment of thirteen hospitalized patients with serious, complicated injuries of the chest wall are reviewed. Two case reports are included.

The results indicate that the relief of pain following fractured ribs, by interrupting the nervous pathways with paravertebral injections of anesthetic solutions is a worthwhile therapeutic measure.

REFERENCES

1. Maingot, R. Textbook of Postgraduate Surgery, p. 4447. New York, 1937. D. Appleton Century Co.
2. ten Horn, C. Fracture of the rib. *Nederl. tÿdscbr. v. geneesk.*, 2: 587, 1921.
3. Haubensak, O. Ueber Rippenfrakturen und ihre Folgezustande; Bearbeitet an Handvon 1156 Fallen der Schweizerischen Unfallversicherungsanstalt aus dem Jahre 1920. *Arcb f. Ortbop. u. Unfall-Cbir.*, 21: 551–594, 1922–1923.
4. Hertzka, E. Fracture of ribs. *Wien. med. Wcbnscbr.*, 70: 336, 1920.
5. Berry, F. B. Wounds of thoracic viscera. *Am. J. Surg.*, 39: 12–17, 1938.
6. Labat, G. Regional Anesthesia, 2nd ed. Philadelphia, 1928. W. B. Saunders & Co.
7. Peterson, M. C. Paravertebral alcohol injection for cardiac pain. *Anesth. & Analg.*, 17: 35–37, 1938.
8. Richardson, E. P., and White, P. D. Sympathectomy in the treatment of angina pectoris. *Am. J. M. Sc.*, 177: 161, 1929.
9. White, J. C. The Autonomic Nervous System. New York, 1935. Macmillan.
10. Latteri, S. Alcoholization of intercostal nerves in therapy. *Riv. san. sicilana*, 21: 249, 1933.
11. Leotta, N. Simplification of technique of alcoholization of intercostal nerve block. *Lotta Contro la tuberc.*, 4: 140, 1933.
12. Grieco, Fausto. Alcohol injection of intercostal nerves in the treatment of fractured ribs. *Riv. de cbir.*, 3: 507, 1937.
13. Rabboni, F. Treatment of fractured ribs by alcohol injection of intercostal nerves. *Cbir. d. org. di movimento*, 22: 263–272, 1936.
14. Zoppi, Bruno. A new method for treatment of rib fractures. *Gior. veneto di sc. med.*, 10: 666, 1936.

BODY SECTION RADIOGRAPHY IN SURGICAL CONDITIONS*

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BODY section radiography is an inclusive term applied to methods of radiographing a selected layer in the body to the exclusion, in greater or lesser degree, of other layers. This type of radiography can be done in several ways, all of which have specific terms,† stratigraphy, planigraphy, tomography, laminagraphy, and serioscopy. It seems desirable to restrict these specific terms to the particular method employed, or to the type of apparatus used, and to employ a general term to cover all of them. For a description of these several methods (except serioscopy), the reader is referred to the articles by Andrews,[1] Kieffer,[2] and Moore.[3] Because the principle is complex, a simple explanation of the fundamental basis of all the methods employed in body section radiography is in order.

If a source of visible light and a recording medium be revolved about a fixed axis, the shadow of any object in this axis will maintain a constant relationship to the light and the recording medium. Any object not directly in the axis, about which revolution takes place, will have a constantly shifting relationship which is dependent on its distance from the axis and its relationship to either the light or the recording medium. This is seen in the diagram. (Fig. 1.) The diagram illustrates the simplest form of movement whereby shadows of objects in alignment can be separated from each other. With combined movements, the separation of shadows can be made more effective.

† Stratigraphy, Vallebona; planigraphy Ziedses des Plantes; tomography, Chaoul and Grossmann; laminagraphy, Moore; serioscopy, Ziedses des Plantes and Cottenot.

The rules which apply to visible light apply with equal force, but with a qualification, to *x*-light. This qualification will be referred to later. In radiography, the *x*-light records the sum of all structures capable of forming a shadow on the recording medium, the radiograph. Many maneuvers have been devised to separate superimposed shadows by changes of position of *x*-ray tube, film and object. All of them are of limited use. The first effort to eliminate undesired shadows by means of a coördinated movement of *x*-ray tube and medium, during exposure, was by Bocage in 1921. The investigations since that time can be found fully described in the references already given.

With visible light, the limitation in the separation of shadows has been referred to above. It is to be remembered, in shifting superimposed shadows with *x*-light, that a proportion of the shadows which are shifted is distributed throughout the path of travel of the beam of *x*-rays because of their penetrability. In consequence, the coördinated movement of *x*-ray tube and its recording medium, in addition to shifting shadows above and below the axis of movement, must also distribute them widely and evenly. The more mathematically harmonious and complete the movement of the *x*-ray tube and film, the better the distribution of the undesired shadows. The merits of the different movements employed in body section radiography need not otherwise be discussed here.

A selected level or layer in the body can be radiographed, to the greater or less exclusion of layers above and below, in three ways: through coördinated, synchro-

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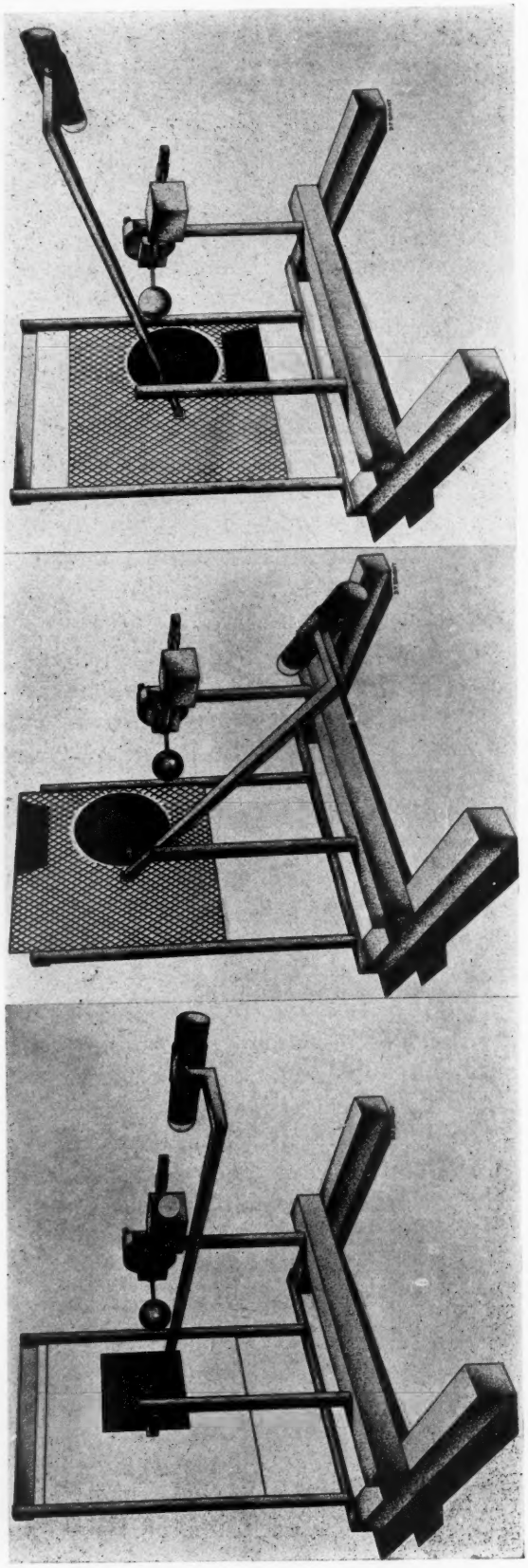


C B A

FIG. 1. A, cube and sphere are in alignment in a beam of light. Shadow of cube obliterates that of sphere on recording medium. B, beam of light and recording medium move on same axis as that of sphere; shadows of cube and sphere are separated. C, reverse of preceding movement.

nized movement in different directions of *x*-ray tube and film during exposure; by keeping the tube and film stationary and revolving the body—in Vallebona's stratigraphy the tube and film remain stationary and the object (the patient) is moved, which is the opposite principle to that in the other methods of body section radiography; and by making multiple radiographs at different angles, the tube, film and body being stationary. In the last case, the radiographs are moved over each other in a special apparatus devised for that purpose, the serioscope. The films are placed in the serioscope in the order in which they have been made. The movement imparted to them is in relationship to the angle at which they were exposed.

In all methods of body section radiography there are definite thicknesses of layer which can be recorded clearly. The thickness varies with the apparatus and the type and extent of excursion of tube and film. The laminagraph has most of the possible practical movements of tube and film, with the means of varying the thickness of the layer to be investigated, the lower limit being about 5 mm. Increasing this decreases clearness. The laminagraph can be used for serioscopy.

It is a general principle that body section radiography has its chief value where there is a maximum number of superimposed structures (Fig. 2) and, conversely, it is of less value where the opposite is the case. Unwanted shadows are displaced and dispersed by this means, but there is also great value in the fact that the shallow depth of layer permits minimal contrasts in density to be recorded on the film. (Fig. 3.) Finer changes in tissue, therefore, that otherwise would be undiscoverable, can be visualized; for example, small areas of calcification in soft tissues or small fragments of fractured bone, or foreign bodies of little density. Body section radiography, therefore, has its chief use in regions or conditions where standard *x*-ray practice is inapplicable.

It is to be borne in mind, however, that the limitations which apply to radiography, because of great size or massiveness of the part to be studied, cannot be overcome by any method of body section radiography and, as a corollary to this, it must be stated that, so far as experience has gone, laminagraphy is an adjunct to, and not a supercession of, conventional methods.

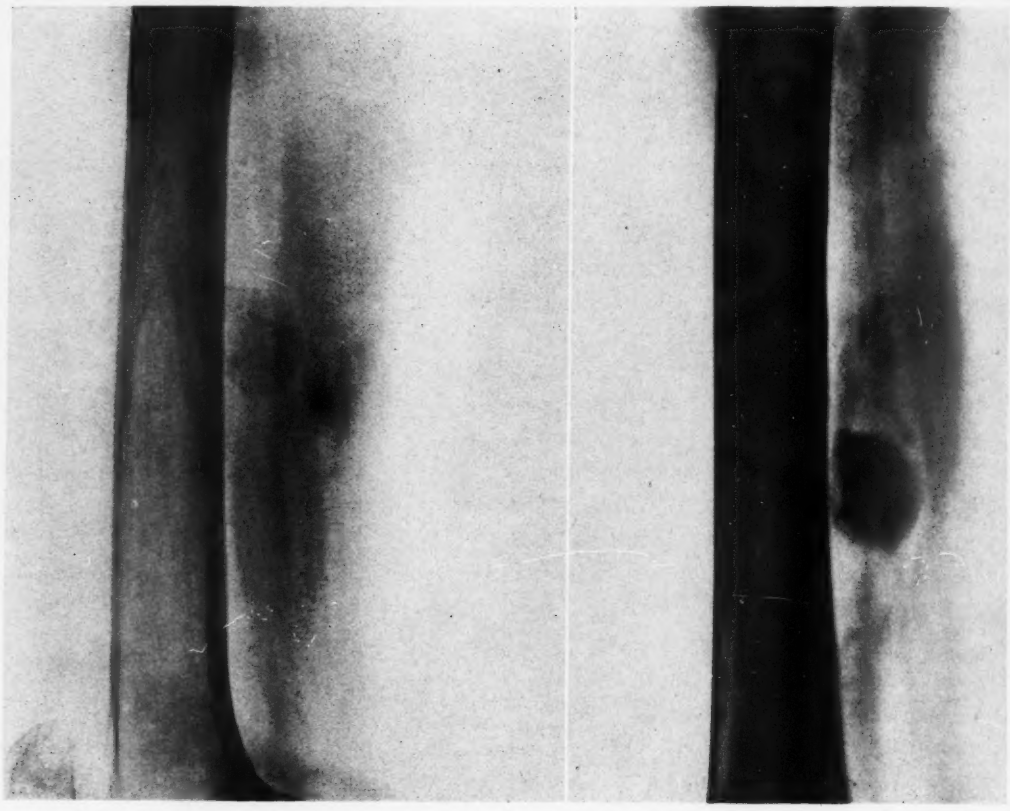
Fig. 2. Laminagraph of lateral view of normal hip joint through opposite hip joint.

RESPIRATORY SYSTEM

Body section radiography is of the utmost value throughout the respiratory tract. This method has more applications in the lower part of the tract than in the upper air passages, for the lower part is subject to more pathologic states which can be investigated advantageously by any type of *x*-ray procedure. It does not promise ever to be very useful in acute or early chronic pulmonary disease. It may demonstrate, however, that the disease is more advanced than was thought to be the case by other methods of examination.

But this is not true in early tracheobronchial disease, as there is great promise

of valuable aid from this method in early disease, especially of an occlusal type. In the normal respiratory tract, the larger bronchial branches and likewise their narrowing and occlusion. In the case of a pulmonary cavity, its depth is accurately



A B

FIG. 3. Tumor of thigh. A, conventional radiograph. B, laminagraph.

divisions of the tracheobronchial tree can be delineated through a large part of their extent by the use of properly exposed serial laminagraphs.

Occasionally the vascular tree can be shown equally well. The presence of cavities (Fig. 4) and, to a lesser extent, of tumors, is discoverable. These cannot be found by any other means. There is every reason to believe that the absence of cavitation can be shown with equal facility, but it must be remembered that there is a certain minimum of contrast in density which must be present. For example, if a cavity is completely filled with fluid contents, it would not be demonstrated in an opaque area of the lung. The thinness of the layer radiographed frequently permits, because of the low density differential, the displacement and distortion of very fine estimated and its third dimension very closely approximated through serial laminagraphs. This fact is invaluable in the surgical therapy of such lesions.

Displacements, distortions and compressions of the tracheobronchial tree are discovered readily. The degree of obliteration of pulmonary cavitation through collapse therapy of any type can be estimated accurately only by this method. Recurrent cavitation in the lung following therapeutic measures can be detected where its presence would be undiscovered by standard methods.

OSSEOUS SYSTEM

The osseous system comes next in this field of usefulness, body section radiography being of particular value about the following portions of the axial skeleton: the

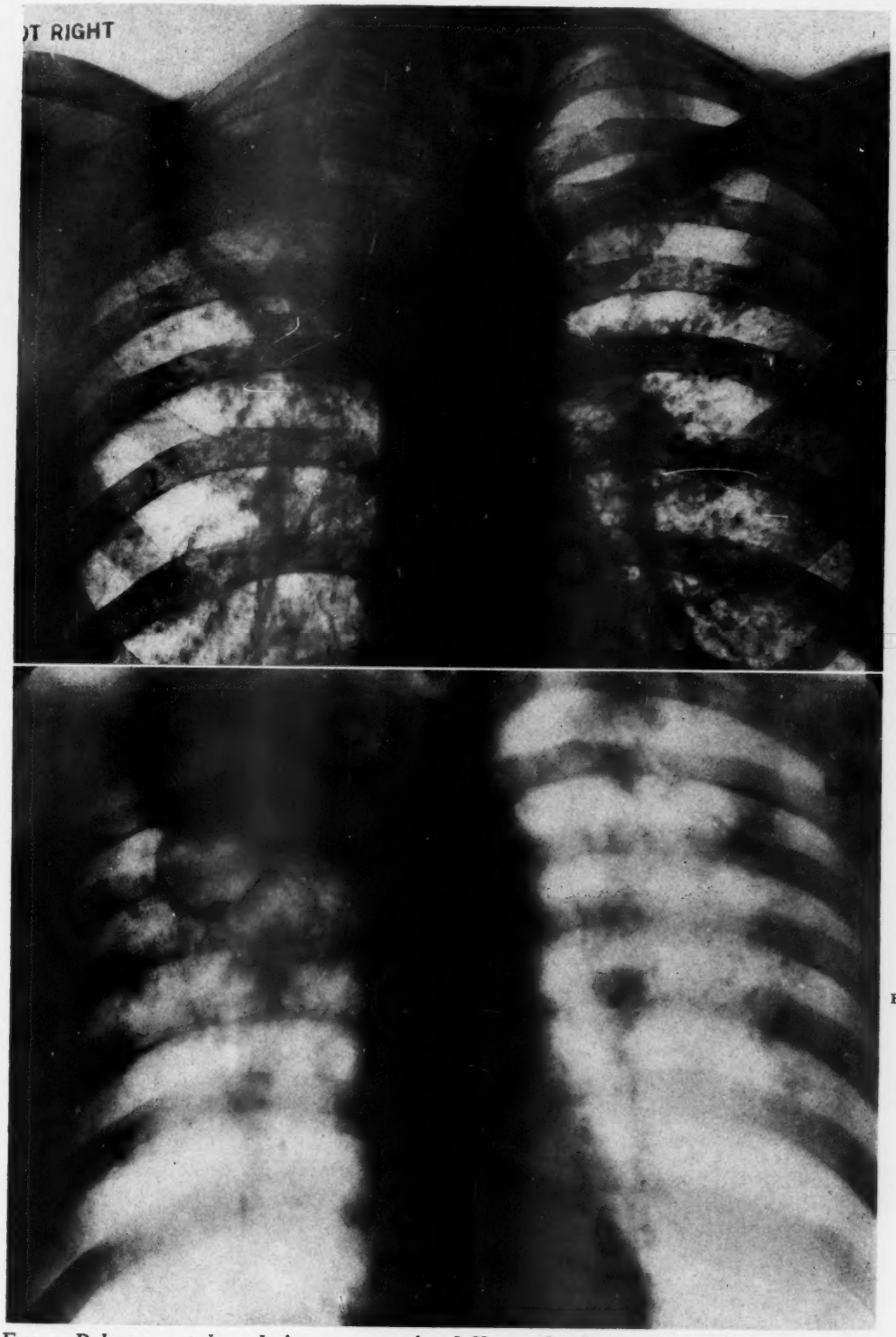
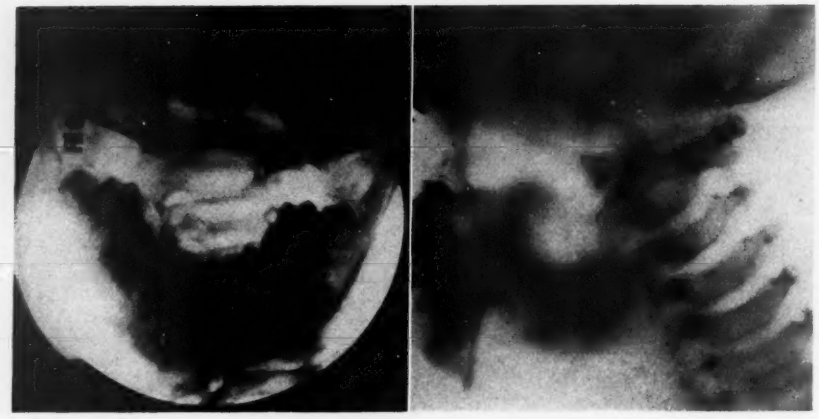


Fig. 4. Pulmonary tuberculosis. A, conventional film. B, laminagraph reveals large activity.

skull, particularly its base, the cervical spine, the dorsal spine, sternum and ribs, in the order given. In the remainder of the radiography they would not be observed. This is equally true of the remainder of the skeleton. In the appendicular skeleton,

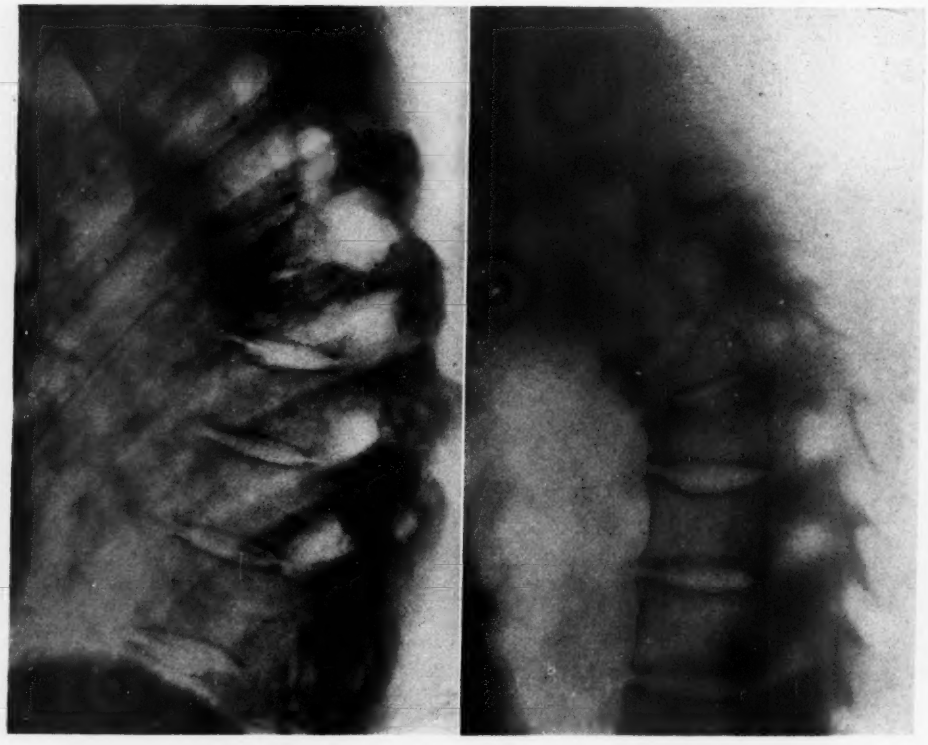


A B

FIG. 5. Torticollis. A, conventional film through open mouth. B, laminagraph reveals rotary dislocation of atlas.

axial skeleton, the lumbar spine and sacrum, this method has yet to prove its superiority over conventional radiography. body section radiography has been of great value in a limited number of instances.

About the base of the skull the greatest



A B

FIG. 6. Old trauma of dorsal vertebrae. A, conventional film. B, laminagraph.

However, shall foci of destruction in bone, either of infectious or neoplastic origin, can be detected readily,although with standard usefulness has been in examination of the temporal bone, particularly in the case of the mandibular articulation. Pathologic

conditions affecting the internal and external auditory meati the existence of which is unsuspected otherwise, can be discovered. Disease and trauma of the occipito-atlantal junction and at the axis (Fig. 5) have been found with the greatest facility, and correct treatment instituted. Laminagraphic examination of the remaining cervical vertebrae has been of less help.

The upper dorsal vertebrae are visualized clearly in the lateral view through both shoulder girdles. At a lower level they are seen free from the shadows of the overlying ribs and lung markings. (Fig. 6.)

Shadows of structures lying posterior to the sternum can be so blurred that this bone is delineated clearly throughout its extent, but in practice separate films usually are required for the manubrium and gladiolus. The presence or absence of trauma or disease of the sternum can be established readily. This is true also of obscure conditions involving the ribs.

In the appendicular skeleton, small defects in bone have been found, as has been mentioned above, after standard methods have failed. In a patient who was thought to have a primary malignant tumor of the bone, the zone of calcification which led to that belief was shown, through laminagraphic examination, to be entirely independent of the bone. Very small avulsion fractures about the joints have been discovered when their presence was not suspected.

NERVOUS SYSTEM AND NEUROVENTRICULOGRAPHY

Except where disease of the nervous system results in bone changes, the laminagraphic examination of the nervous system has not been of value, but the possibilities which lie in the small density differential of the thin layer may be useful in the future. In investigation of intracranial calcifications and in examination of the sella turcica, no superiority over standard methods has been found.

This method has not been employed in either neuroventriculography or encephalography. However, its employment in this type of examination has its advocates.

DIGESTIVE AND URINARY SYSTEMS

Body section radiography has not been used, except experimentally, in the digestive and urinary systems. However, in both systems there is the possibility of material aid in cases in which the recording of slight contrasts in density would be helpful, or in which elimination of the shadows of unusual objects is desired.

VASCULAR SYSTEM

In the vascular system, calcifications in aneurysms have been found and the sac outlined.

There is a possibility that the smallest intracardiac calcifications, since their exact depth in the body is known, can be found, and it is probable that there will be important developments in this field of investigation.

In this connection it may be said that like possibility exists in the other soft tissues. Exact localization of foreign bodies is possible in serial laminagraphs.

TECHNIQUE

It is to be repeated that body section radiography is not to be substituted for standard *x*-ray methods. The latter should precede always, and they are a valuable guide in determining the extent of the area to be examined and the level at which examination should begin.

Only general statements can be made as to the technique. Knowledge as to type and amplitude of movement, and thickness of layer to be radiographed, has to be gained by experience. The general rules of radiography, in regard to the position of the patient, voltage, current, and time of exposure, apply in body section radiography approximately the same as in standard methods. The technique is not difficult, but still requires a considerable amount of trial and error.

SUMMARY AND CONCLUSIONS

Body section radiography has been defined, and a simple exposition of the fundamental principle of all methods of body section radiography has been made. The several methods of carrying out this procedure have been briefly defined and described. The possible clinical applications of body section radiography in the several anatomical systems have been briefly discussed.

It is concluded that this method of radiographic examination holds great promise for extending the value of radiography.

REFERENCES

1. Andrews, J. R. Planigraphy. 1. Introduction and history. *Am. J. Roentgenol.*, 36: 575–587, 1936.
2. Kieffer, J. The Laminagraph and its variations. *Am. J. Roentgenol.*, 39: 497–513, 1938.
3. Moore, S. Body section roentgenography with the laminagraph. *Am. J. Roentgenol.*, 39: 514–522, 1938.

Physical pain is certainly influenced by a *mental factor*, by energy, by freewill. But how do these factors take effect? Do they act only as a brake on the expression, the representation of pain? Or are they able actually to diminish the acuteness of our painful perception?
From—"Surgery of Pain" by René Leriche (Williams & Wilkins).

INCIDENCE OF HEADACHE, NAUSEA AND VOMITING FOLLOWING SPINAL ANESTHESIA

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SINCE the introduction of spinal anesthesia by Corning[1] in 1885 the literature has contained many reports concerning its advantages and disadvantages. Its usefulness has been definitely established by such pioneers in this field as Bier of Germany, Tuffier of France, Babcock, Pitkin, Stout and others in America, and it is now widely recognized as a valuable addition to the surgeon's armamentarium. The technique of administration has been well standardized, with close adherence to certain basic principles which these men have emphasized. Numerous additions and modifications have been described and various needles and solutions perfected, but analysis has demonstrated that the method of lumbar anesthesia is as safe as its operator. Babcock[2] states, "No solution or technical innovation has displaced the necessity for an accurate technique and rigid supervision. The dangers of spinal anesthesia remain largely with the user rather than the drug or technique employed."

The present analysis is based upon work by two members of our hospital staff, each using the same instruments, solutions and the same technical procedure. The purpose of our study was to determine as accurately as possible the incidence of such post-lumbar anesthetic complications as headache, nausea, and vomiting as found in a series of 500 patients, the majority of whom had been subjected to rectal operations. This selection of cases was made because of the infrequency of these complications, under ordinary conditions, with this type of surgery. Most of the patients in the series analyzed were ambulatory twenty-four to forty-eight postoperative. Since such operative procedures are attended by little shock and by little disturbance to the gastrointestinal tract, it was thought that a more accurate appraisal of the postoperative effect of the anesthesia would be obtained. Most of the studies reported in the literature concerning headache and vomiting following spinal anesthesia have been made on patients subjected to abdominal operations. It is the rule that such patients are recumbent for a period of at least seven to twelve days. This may explain the discrepancy between our figures regarding the frequency of headache and those to be found in the majority of reports. The experience of Harrison[3] with spinal anesthesia among the natives of Arabia, where existing conditions made it necessary that his patients be ambulatory almost immediately following surgery, tends to support this contention. Parker's[4] report indicating a 20 per cent incidence of headache following lumbar puncture alone furnishes additional evidence that the frequency of headaches is much higher where the period of recumbency is short.

Our study was based upon an analysis of 500 cases of lumbar anesthesia in which novocaine crystals were used. In this series 453 patients were given less than 100 mg. of novocaine and forty-seven had 100 mg. or more. The technique described by Labat was observed in at least 90 per cent of the cases. The headaches were classified as moderate or severe. The severe type included all patients who suffered intense pain and those who complained of headache for a period of more than twenty-four hours. In all, 153 patients suffered from postanesthetic headache; of these sixty-seven (13.4 per cent), were considered as severe and eighty-six (17.2 per cent), as

moderate, for a total incidence of 30.6 per cent. In 143 cases (93 per cent), the onset occurred within twenty-four hours after the lumbar puncture; only in ten cases (7 per cent), did the onset occur after twenty-four hours. It was also noted that of the 453 patients who had less than 100 mg. of novocaine, 29.6 per cent had headaches, as compared to 10 per cent of the forty-seven patients who had 100 mg. or more. One patient suffered almost continuous headache for seven days and has complained of frequent headaches for a period of eight months.

When compared with similar figures of other investigators a wide variation was noted. (Table 1.) However, this might be

TABLE 1
HEADACHES FOLLOWING LUMBAR ANESTHESIA

	No. of Cases	Incidence of Headaches, Per Cent
Evanston Hospital	500	30.6
Pennsylvania Hospital[8]	1500	7.3
Babcock (1913)[9]	5000	21.0
Koster-Weintrob[10]	6000	10.0
Broglio[11]	1146	12.5
Campbell[12]	410	3.0
Wells[6]	557	2.5
Overholt-Muller[7]	533	4.0
Stein-Tovell[13]	150	6.6
HEADACHES FOLLOWING LUMBAR PUNCTURE ALONE		
Parker[4]	200	20.0

expected when it is considered that most series of postanesthetic complications to be found in the literature represent a wide variety of surgical conditions. Inasmuch as the character of the surgical procedure may greatly influence the postoperative course it was our belief that a series of cases subjected to the same, or closely related, operation would provide a more accurate study.

With the possible exception of the one young woman who has complained of frequent headaches and occasional spells of "faintness" for a period of eight months there have been no serious after effects among any of the patients of our series. A neurological consultant, after examining this patient, assured us that no evidence was present which suggested any relationship between the complaints and the spinal anesthesia, but a history which definitely dates back to the time of operation seems more than coincidental.

It has been our observation that the most satisfactory treatment of a post-anesthetic headache is a prolongation of the "flat in bed" period. Nelson[5] has shown that the cause of the post-lumbar headache was the leakage of spinal fluid from the canal after the puncture and that the incidence of headache can be reduced substantially by plugging the puncture wound with catgut. He also found that the supine position seemed to relieve the headache much more satisfactorily than any other form of treatment. Harrison[3] believes that the injection of irritating solutions

TABLE II
INCIDENCE OF NAUSEA AND VOMITING FOLLOWING LUMBAR ANESTHESIA

	Nausea and Vomiting, Per Cent	Nausea, Per Cent	Vomiting, Per Cent
Evanston Hospital	34.6		
Pennsylvania Hospital[8]	7.3		
Wells[6]	50.0		
Campbell[12]	2.0		
Overholt-Muller[7]	....	31.0	14.0
Broglio[11]	....	18.0	13.0

plus the increased absorption of cerebrospinal fluid at the site of irritation are the factors responsible for most headaches. He found a 5.48 per cent solution of procaine HCl to which $CACl_2$ is added in sufficient quantity to make its strength 0.024 per cent, to be least irritating. He also advocates intravenous administration of 5 per cent dextrose solution in normal saline at the conclusion of the operation, believing that this obviates the danger of

disturbed hydrodynamics of the cerebrospinal system.

Nausea and vomiting were encountered in our series with greater frequency than that reported by most operators. (Table II.) However, Wells[6] records 50 per cent as the proportion of his patients who suffered nausea and vomiting, while Averholt and Muller found that 31 per cent of their patients had experienced nausea, 14 per cent of them having vomited. Most of our patients received morphine preliminary to subdural anesthesia which may have accounted for some of the nausea, but a lack of control cases renders any conclusions in this regard unreliable. The fact that most of the patients in our series underwent operations on the lower bowel undoubtedly was a factor in the high incidence of nausea.

Twenty-one per cent of the patients studied required catheterization. However, as temporary bladder dysfunction is common following rectal surgery with all types of anesthesia this finding was not considered worthy of comparative analysis.

Despite the frequency of headaches, nausea and vomiting following spinal anesthesia, its wide adoption is testimonial to its usefulness. That it confers certain benefits upon both the patient and the surgeon which can be obtained with almost no other form of anesthesia is admitted by most authorities. We have chosen this method of anesthesia for anorectal surgical patients because of its relatively simple administration and because it provides relaxation of the anal sphincter which is superior to all other forms of safe anesthesia, except caudal-sacral block.

On the other hand, while the disadvantages of lumber anesthesia for surgical procedures confined to the abdomen and lower extremities are few, certain minor complications are both frequent and distressing, and of these, headaches deserve first consideration. It is our opinion that headaches occur as a complication of spinal anesthesia much more frequently than is commonly realized and that their incidence is inversely proportional to the time of recumbency. Such a state is deserving of further investigation if we are to continue the use of this anesthetic agent for comparatively minor surgical procedures. We have adopted Harrison's proposal of administering dextrose solution by the intravenous route in an effort to reduce the number of headaches, but as yet our series of patients so treated is too small to deserve comment.

SUMMARY

A report of 500 cases of lumbar anesthesia is submitted with special reference to the incidence of postanesthetic headache, nausea, and vomiting in patients who are ambulatory within a short period after surgery.

1. Postanesthetic headache occurred in 30.6 per cent of the cases; this is a much higher incidence than one finds in most published reports and may be explained upon the basis of the following considerations: cases reported in the literature are not selected cases, but those usually requiring seven to ten days of postoperative bed rest; the cases analyzed in the present study were largely those on whom anorectal operations had been performed, these patients requiring only a short period of recumbency.

2. The dosage of spinal anesthesia apparently has no bearing upon the incidence of post-lumbar anesthesia headaches.

3. The onset of the postanesthetic headache usually occurs within twenty-four hours after the lumbar puncture.

4. The most effective treatment of such headaches is prolongation of the period of bed rest.

5. Nausea and vomiting occurred in 34.6 per cent of the patients following lumbar puncture anesthesia.

REFERENCES

1. Corning, J. L. Spinal anesthesia and local medication of the cord. *New York J. Med.*, 42: 483–485 (Oct. 31) 1885.
2. Babcock, W. W. Spinal anesthesia in fact and fancy. *Surg., Gynec., & Obst.*, 59: 94–100 (July) 1934.

3. Harrison, P. W. Postanesthetic headache. *Arch. Surg.*, 32: 99–108, 1936.
4. Parker, H. L. Headache following diagnostic spinal puncture. *Proc. Staff Meet. Mayo Clin.*, 4: 369 (Dec. 18) 1929.
5. Nelson, M. O. Post-puncture headaches. *Arch. Dermat. & Syph.*, 21: 615–627 (April) 1930.
6. Wells, J. R. Spinal anesthesia with anhydrous cocaine; observations on 557 additional cases. *Ann. Surg.*, 85: 757–764, 1927.
7. Overholt, R. H., and Muller, G. P. Spinal anesthesia. *Ann. Surg.*, 94: 738–750 (Oct.) 1931.
8. King, O. C. Spinal analgesia. *Ann. Surg.*, 101: 690–701 (Feb.) 1935.
9. Babcock, W. W. The dangers and disadvantages of spinal anesthesia. *New York J. Med.*, 98: 897–903, 1913.
10. Koster, H., and Weintrob, M. Complications of spinal anesthesia.
11. Broglio, R. Spinal anesthesia in series of over 1000 cases. *J. A. M. A.*, 87: 794, 1926.
12. Campbell, M. F. Spinal anesthesia. *Ann. Surg.*, 84: 42–50, 1926.
13. Stein, J. J., and Tovell, R. M. Spinal anesthesia. *Am. J. Surg.*, 30: 282–286, 1935.

It appears that hypophysial deficiency probably is accompanied by increased catabolism of proteins of endogenous origin and imperfect utilization of proteins of exogenous origin, whereas anterior pituitary extract favors protein synthesis.

From—"The Physiology and Pharmacology of the Pituitary Body," vol. II, by H. B. Van Dyke (University of Chicago Press).

THE SURGICAL PROBLEM IN PERIPHERAL VASCULAR DISEASE*

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THE surgical treatment of peripheral vascular disease differs radically from the general surgical therapy of similar problems in non-vascular patients and lack of understanding of this differentiation leads frequently to serious and unnecessary complications. With the modern care of the arteriosclerotic and diabetic patients and the improved results in those with thrombo-angiitis obliterans, more of these patients are reaching the age when surgical complications will arise. A report of the surgical experience of the Vascular Clinic at New York Post-Graduate Hospital is therefore timely.

The problem is handled jointly on a combined service with attention directed to the medical and the surgical sides at the same time. The importance of this factor has not been sufficiently stressed. While our purpose is to detail the surgical management of the vascular problems, we wish to emphasize that neglect of the medical or conservative therapeutic measures will result in ultimate failure, no matter how carefully the surgical part is conducted. We feel that only by such synchronized care can satisfactory results be achieved.

SURGICAL TREATMENT OF INFECTIONS IN VASCULAR DISEASE

1. *Infections.* An abscess, or paronychia, in the vascular patient requires earlier but less radical intervention than in other types of patients, depending, of course, on the extent of arterial disease. Poor vascularity means poor tissue resistance, and while early incision is advantageous, the restricted blood supply will retard healing in any extended opening or exploration.

Anesthesia is a problem in these patients. An agent which anesthetizes by infiltrating the tissue and constricting the already restricted vessels may cause great damage to the tissues. Figure 1 shows the effect of a simple novacaine block at the base of the toe in a patient with previously undiagnosed thrombo-angiitis obliterans surgically treated for an infected ingrown toe-nail. The circulatory status of the foot was not determined prior to operation. The dorsalis pedis artery was occluded and simple palpation probably would have avoided this complication. Adrenalin, frequently added to a local anesthetic, has a more deleterious effect on the tissue by its vasoconstrictor action and must be entirely avoided. Such anesthetics as ethyl chloride, which "freeze" the tissues, are dangerous for the same reason. The importance of feeling for the dorsalis pedis and posterior tibial pulsations prior to contemplated foot surgery is to be emphasized.

2. *Cellulitis.* In the diabetic patient, earlier and more radical intervention is necessary than in the other groups, as the diabetic is non-resistant to slight infection. In the arteriosclerotic and thrombo-angiitis obliterans groups the incision may be much less extensive, as the infection frequently will localize, and even tissue with a necrotic appearance at times will revitalize following the stimulation of collateral circulation by conservative means. Thus early and conservative intervention is the rule, unless the patient has a diabetic complication. Frequent hot soaks

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followed by dry dressings, as will be described, aid materially.

3. *Tenosynovitis.* It is important in each infection of this type to *x*-ray for bone involvement. The usual tenosynovitis in vascular patients is of the slow, necrotizing types which destroys sections of the tendon progressively. Adequate opening of the overlying tissues and the tendon sheath is necessary, but necrotic areas in the tendons should not be excised. Such excision not only destroys portions of the tendon, which may resolve, but an excised tendon will retract, setting up a new site of necrosis some distance up the sheath. The sloughing tendon is permitted to demarcate and during this time serves as an excellent drain. The substitution of some foreign body for such a natural drain is surgically unsound. Increasing the general resistance, with care as to the vascular and metabolic problem, the correction of anemia or other pathologic states, combined with the prevention of any local pocketing in the wound, will facilitate the recovery in these conditions.

4. *Osteitis.* This condition results from the general avascularity and disuse. Unless this cause is recognized, an amputation may be performed on the erroneous *x*-ray diagnosis of osteomyelitis. Figure 2 shows the type of avascular osteitis frequently seen in these patients. Such bones will regenerate when the overlying infection subsides. Any associated infection with the osteitis necessarily must be drained.

5. *Osteomyelitis.* Osteomyelitis, as shown in Figure 2, in the fifth metatarsal phalangeal area requires sacrifice of the part involved. This will be more carefully considered with amputation.

Fig. 1. Gangrene of the great toe following novocaine infiltration at the base for drainage of infected ingrown nail. (Thrombo-angiitis obliterans.)

6. *Gangrene.* Unfortunately, we know of no optimal time to amputate. Observation of regeneration of an apparently lost part makes us conservative. In the diabetic patient, it is not wise to procrastinate too long as an optimal amputation time cannot be maintained for any great period. A study of the blood supply should be made by palpation, oscillmetric readings and at times arteriography with a Roentgen opaque solution. Attempts to conserve the extremity below the place shown by these tests to be the point of inadequate circulation only result in multiple amputations, with increased risk. Delay in the arteriosclerotic type is safe for a time if there is no evidence of extension of the infection, above the ankle. Active medical therapy frequently results in surprising recovery. When amputation is performed, it means mid-thigh, except in the very rare instance, since adequate circulation is rarely demonstrable below the knee in these advanced cases.

Patients with thrombo-angiitis obliterans require local amputations late and only after a thorough demarcation has occurred. Conservative therapy is so successful with these patients that we are glad to report that in five years there has only been one major amputation in this disease on the

Vascular Service, with its very active service. Recently we have employed spinal anesthesia which temporarily paralyzes the sympathetics, as a therapeutic measure in threatened gangrene. Embolic gangrene requires high amputation in one-half of those patients surviving the original vessel insult.

Amputations for vascular disease differ from those for traumatic injuries due, of course, to the poor blood supply. Oscillometric readings are of utmost importance in this respect as a guide to the extent of the vessel patency. Figure 3 shows a comparison of the oscillometric readings, the arteriographic tracings, the clinical data, and the pathologic specimen in a patient with embolic gangrene. We were able to demonstrate the fact that the blood supply was inadequate well above the actual arterial block with our oscillometric readings, and we rely more and more on these simple and effective determinations.

The general preparations for amputations include enemas, control of any glycosuria and sedatives, dependent on the age and the condition. In addition to this routine, woollen underwear is used, as most of these individuals stand cold poorly. Cyclopropane, if short and light, has been demonstrated to be the least shocking of the inhalation anesthesias for this type of operation, and hyperventilation for some time postoperatively has materially reduced the incidence of pulmonary complications. The medical management must not be neglected at this stage. The effort to increase collateral circulation as by the use of adrenalin neutralizing tissue extracts, typhoid vaccine intravenously, the oscillating bed and thermostatically controlled heat, should be continued up to the amputation time and resumed thereafter. The problem of the use of prophylactic gas gangrene serum is an individual one, but in the aged, where there has been fecal contamination, it is recommended.

Local preparations are important because of the skin contaminations from incontinence and long bed confinement. The skin is cleansed with green soap the day before the operation and twice the operative day.

Fig. 2. Generalized avascular osteitis well demonstrated with an osteomyelitis in the fifth metatarsal phalangeal joint area. Note differentiation of avascular osteitis with its generalized lime absorption and osteomyelitis with bone cortex and continuity lost.

Iodine, if used, must be very weak and removed with alcohol at once to prevent damage to the non-resistant skin. The tourniquet and Esmarch constrictor should be eliminated as this constriction may injure the intima of the proximal vessels. Skin preparations and drapes should be prepared before induction of the anesthesia to shorten the anesthesia time.

A NEW TECHNIQUE FOR VASCULAR AMPUTATIONS

The accepted mid-thigh amputation techniques have been found to be unsatis-

factory for vascular work. The damaged arterial supply makes the construction of muscle, fascial and skin flaps impossible and such experience in our clinic has led us to the development of this amputation procedure. A simple circular incision is made through all structures down to the bone, without efforts to construct flaps of muscle or fascia. Muscles are divided high with as little trauma as possible with the sharp scalpel, and the large vessels are ligated individually with a transfixion suture. The sciatic nerve is divided on tension, ligated, and after cartwheel injection with absolute alcohol to prevent neuromata, is permitted to retract. The tissues are retracted up the femur, and, without stripping the periosteum, the femur is divided with a hand saw and the rough edges smoothed with a wood rasp. All bleeding is carefully controlled. The wound is closed by uniting the skin and subcutaneous fascia only, with interrupted alloy steel wire, size 34 and 36, without drainage.

The dressings are applied after painting with compound tincture of benzoin and carefully sealed with adhesive tape. Skin extension to remove the pressure at the suture line is secured by modified Buck's extension. This is applied with two inch adhesive strips extending from the upper thigh over a board splint applied on the side of the leg, as advocated by Carter, with the painting of the entire area with waterproof shellac. The elimination of the muscle and fascia closure not only simplifies the technique but, by reducing the usual serum collections, makes a better stump for the attachment of the artificial limb. The thick, mushy stumps so frequently seen are the results of the attachment of a muscle shield over the bone end. The steel wire sutures, presented by Babcock, eliminate the tissue reactions to the suture materials and can be left indefinitely should there by any stump infection.

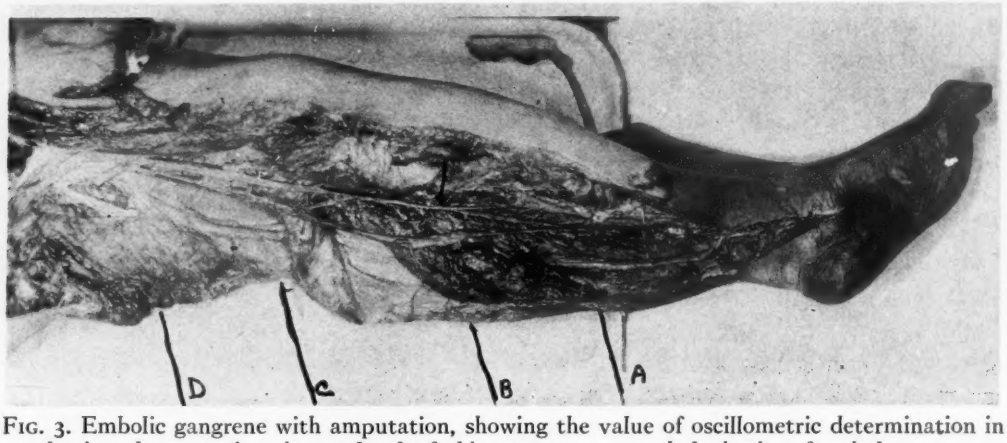

FIG. 3. Embolic gangrene with amputation, showing the value of oscillometric determination in selecting the operative site. A, level of skin gangrene. B, pathologic site of embolus. C, *x*-ray evidence of collateral circulation. D, oscillometric and operative evidence of circulation competency.

With this simplified technique we have been able to effect primary union in nine out of ten patients. We wish to emphasize:

(1) Choice of amputation site by oscillometric readings.

(2) Avoidance of constricting tourniquet.

(3) Elimination of muscle and skin flaps.

(4) Modified treatment of the femur, nerve and vessels.

(5) Closure with interrupted alloy steel wire.

(6) Sealing of dressing.

(7) Adhesive tape extension over splint.

(8) The technical simplicity and speed of the operation.

Should there be questionable vascularity at the operative site, as after emboli, the stump may be left wide open and closed by a secondary plastic repair.

In *local amputations* of toes for arteriosclerosis, a simple disarticulation is used without excision of the tendons, which are left long as drains. In general the toes should be allowed to self-amputate in thrombo-angiitis obliterans cases. Where a clean closure is expected, the racquet type of incision is the best.

Dressings. The dressings of vascular patients differ in certain respects from normal surgical methods. More frequent dressings and a more rigid aseptic technique are required. While wet dressings have an important place in the treatment of general surgical infections, vascular patients stand them very poorly, due to the unavoidable cooling and resultant vasoconstriction and reduction of tissue nutrition. This chilling may be sufficient to change a local condition to a progressively general one. Hot soaks followed by immediate change to dry dressings are used entirely in these infections. The soaking solution must be mild in order not to injure the delicate tissues; such solutions as saline, boric acid or azochloramid in triacetin, 1:500 solution, being effective. The maintenance of the normal temperature is important and the light cage equipped with a thermometer with a constant temperature of from 90 to 95 degrees is used. Such simple measures as the care of the frequently associated fungus infections and the avoidance of moisture between the toes all play their part, together with the medical régime, diabetic control and the elimination of tobacco.

SYMPATHECTOMY

The published results from sympathectomy in the past are not born out by observations and it is our practice to reserve this more serious surgical procedure for Raynaud's disease and the small group of cases which do not respond to other measures. Many of the Raynaud cases, selected instances of thrombo-angiitis obliterans, some classified as essential hypertension and the occasional vegetative neuroses seem to respond fairly well. It is important to realize, however, that in a sympathectomy we are merely treating a symptom and not the cause, and while we are eliminating certain conditions pathologic for the patient's present state, we are substituting other, non-physiologic effects for which there must be compensation. We do not deride the good results achieved by ganglionectomy, for the work of White, Adson, Dandy and others have been authoritative, but we do believe that the entire story as concerns sympathectomy results is far from complete or even understood. For the present this type of procedure must still be regarded as experimental in each case.

The posterior approach to the upper thoracic ganglia has proved satisfactory in *upper extremity ganglionectomy*. By excision of a section of the second rib and second transverse process, the lower cervical, first, second and third as well as part of the fourth dorsal ganglia can be resected.

The lumbar sympathectomy requires general surgical as well as neural or vascular surgical ability, as all the intra-abdominal complications, at times, may be met. Adhesions, growth, etc., may be encountered and the patient will be as subject to the possibility of peritonitis or obstruction as after any laparotomy. The results in general are better than after cervical ganglionectomy.

The incision should be adequate and midline from the pubis to the umbilicus. The intestines are packed off with pads and by Trendelenburg position. On the *left side* the sigmoid and descending colon must be elevated, the posterior peritoneum incised and the ureter, sigmoid and aorta gently retracted medially. The ganglia lying on the lumbar vertebrae medial to the psoas muscle are dissected free by a gauze sponge. The ganglia of the third and fourth lumbar

segments are removed. On the *right side* the vena cava presents an added problem and the incision in the posterior peritoneum should be made laterally to this organ. The vena cava is then retracted medially and by retracting the cecum, small intestine and ureter laterally, the ganglia can be excised. The usual abdominal closure is made after suturing of the posterior peritoneum.

SUMMARY

1. Variations in the treatment of infections in vascular patients from general surgical patients are outlined.
2. General and surgical vascular problems are noted.
3. A new modified technique for mid-thigh amputation is detailed.
4. Sympathectomy is discussed.

THE form of an aneurysm depends first upon whether a considerable stretch of arterial wall gives way or whether one particular spot weakens. A diffuse weakening makes a fusiform aneurysm: a local one, a saccular aneurysm.
From—"Circulatory Diseases of the Extremities" by John Homans (Macmillan).

SELECTIVE THORACOPLASTY

RESULTS OF A SERIES OF THIRTY CASES

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IN the surgical management of pulmonary tuberculosis, many advances have been recently made. The reports of thoracic surgeons have improved the outlook to such an extent that we may now safely promise approximately 85 per cent cures. Of all the patients that the phthisiologist sees, only 15 per cent become and remain well after sanatorium regimen alone. In the remaining 70 per cent, collapse therapy must be instituted. Artificial pneumothorax, while not always our first choice, must be accepted as being the most universally applicable. Unfortunately, due to unsurmountable adhesions, one is not always able to induce a collapse by this method. Surgical collapse then becomes necessary for the desired cure.

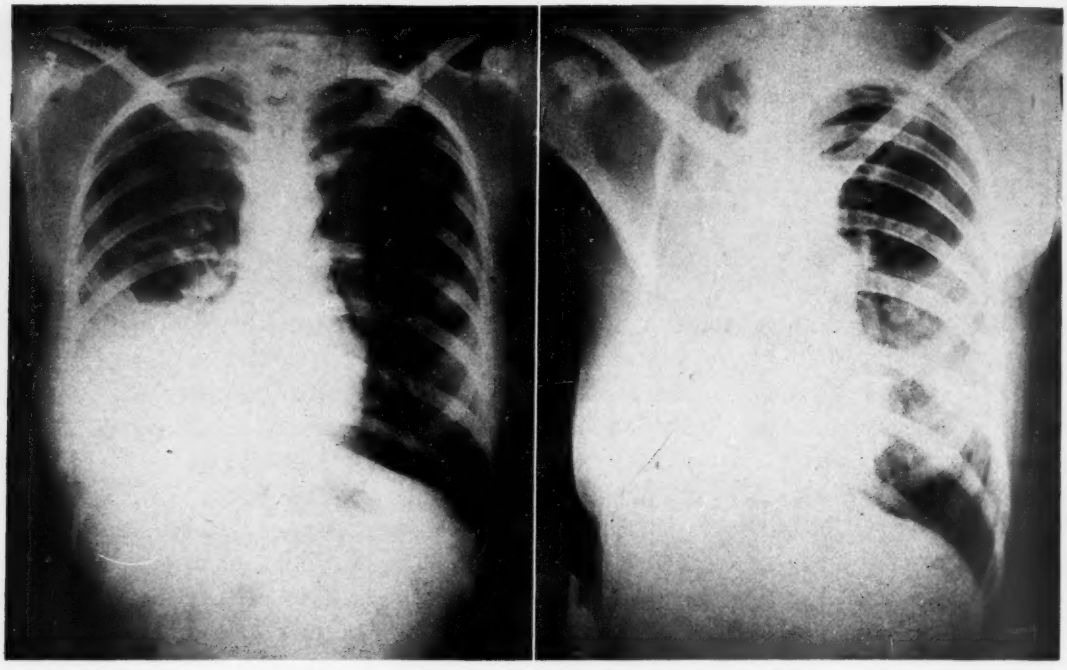
While the indications for surgery in pulmonary tuberculosis have been somewhat limited in years past, there come to our attention more and more instances where some sort of permanent collapse is advisable. Each thoracic surgeon in the field has his own requisites for operation, but we should like to classify these indications in what seems to us a practical and simple manner:

1. Persistence of symptoms which are distressing to the patient; i.e., severe cough, productive sputum, chills, fever, hemoptysis.
2. Persistence of positive sputum.
3. Persistence of *x*-ray evidence of cavitation or honeycombing.

As we see more patients with old smouldering lesions, we have been more and more suspicious that honeycombing is the underlying pathology. While it is not always possible to demonstrate this with ordinary films, we feel that one is more apt to have success with Oechsli's method of taking a hard film with the Potter-Bucky diaphragm. In no instance do we advise surgery for exudative tuberculosis; such a lesion, if not improved with sanatorium care and recumbency, will only glorify mortality statistics. Needless to say, any degree of cavitation must be closed to prevent spread of the disease to other parts of the lung; persistently positive sputum and hemoptysis can frequently be traced to cavitation.

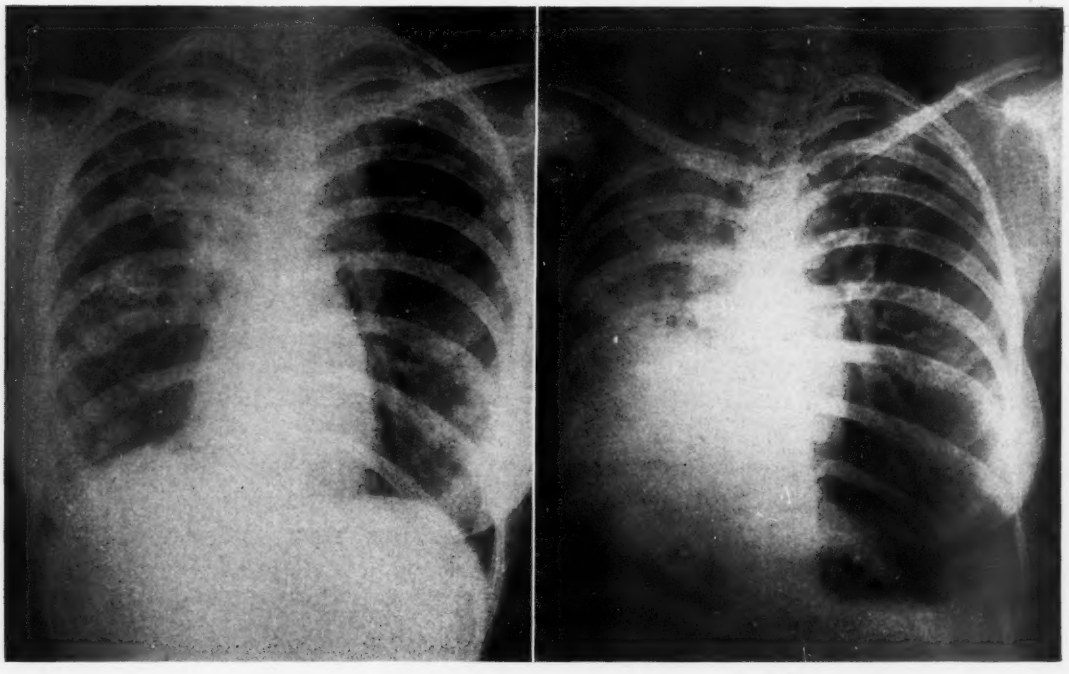
We have no special prerequisites for surgical risk other than our aforementioned maxims. The patient must be as reasonable a risk for thoracic surgery as for any other major surgical procedure. In diabetics, acidosis must be combated; in cardiacs, decompensation corrected; in syphilitics, serology controlled. In fact, anything is done to improve the general operative risk.

Technique, we believe, has been well standardized and needs little comment. Rib beds are routinely formalinized to obviate threatened rib regeneration should a long interval be deemed necessary between stages. No more than three or four ribs are resected at one stage as a rule, the number depending upon the condition of the patient at the operating table. Any evidence of paradoxical respiration terminates the operation. The first rib is removed in toto, and the lengths of succeeding ribs removed is determined by the underlying pathology. In all cases, the resection begins posteriorly with the costovertebral articulation. The last rib or two is resected shorter to afford a better cosmetic appearance to the thoracic cage. In addition, we believe that this helps to prevent impingement of the scapula behind the last resected rib.



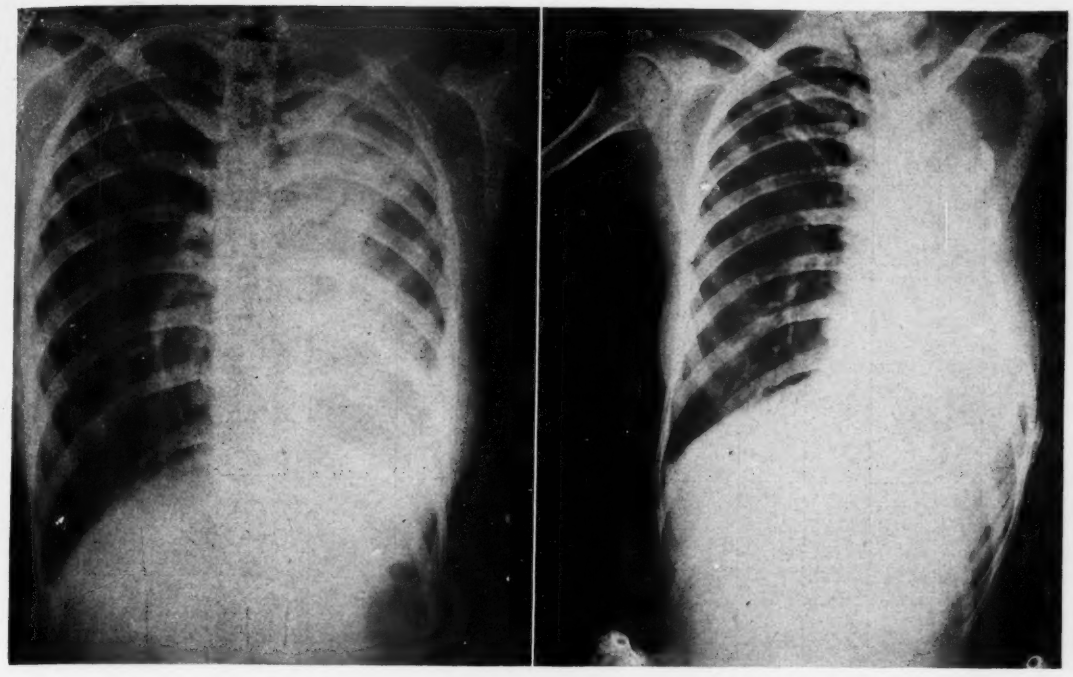
A B

FIG. 1. Case I. A, large basal cavity remaining after temporary phrenic interruption. B, after eleven-rib graded collapse.



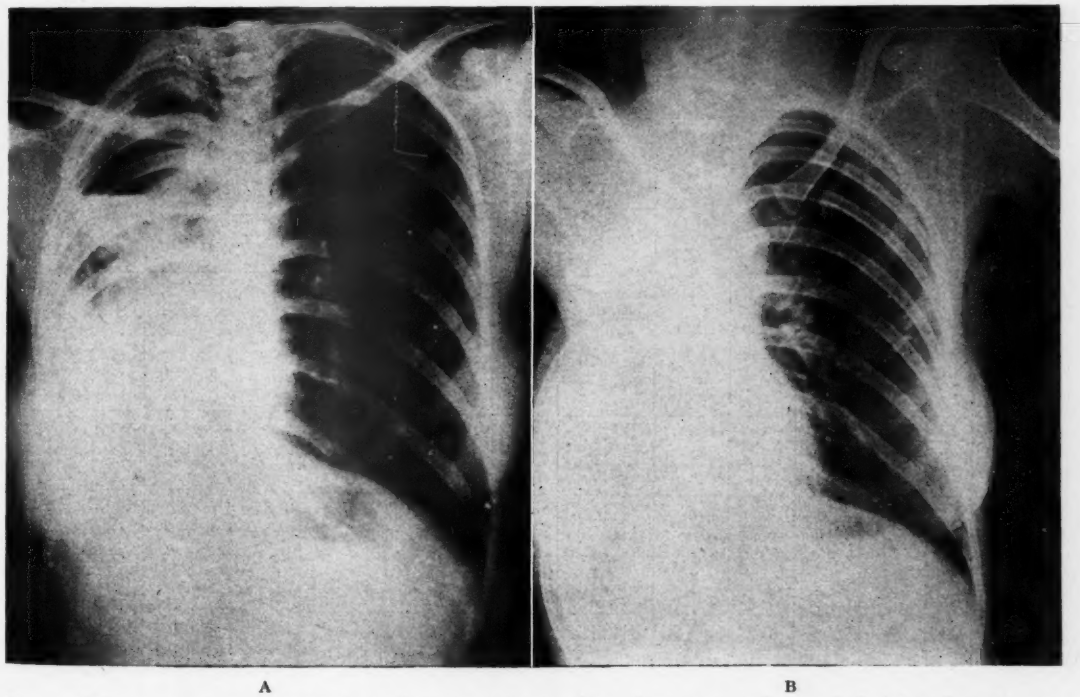
A B

FIG. 2. Case II. A, extensive tuberculosis of right lung and moderate contralateral involvement. B, showing what a phrenic interruption plus one stage of a five-rib collapse will accomplish. Note clearing of opposite lung.



A B

Fig. 3. Case III. A, re-expanded pneumothorax, leaving cavity in upper left lobe. B, after a seven-rib graded collapse.



A B

Fig. 4. Case IV. A, 12 cm. cavity in right upper lobe. B, after four stages of a graded eight-rib collapse.

No.	Case	Sex	Race	Age	Side	Stages	Stage Interval	No. Rlbs	Prelim. Treatment	Duration of Symptoms	Surgical Indication	Remarks
1	H.W.F.	F	W	61	L	1	.....	4	Pneumo.* 3 mo.	6 yrs.	Pos. sputum. X-ray—apical cavity	Markedly improved since 2-4-37. Further surgery refused. Neg. sputum
2	R.J.	M	W	20	L	2	30 days	5	Phren.† 6 wks.	3 yrs.	Pos. sputum. X-ray—apical cavity	Well since 5-20-37. Neg. sputum and x-rays
3	L.M.	M	W	60	R	1	.....	3	San'm‡ 6 yrs.	6 yrs.	Pos. sputum. X-ray—extensive involv. whole rt. with cavity	Incomplete and untraced. Did not return
4	C.E.	M	W	29	L	3	24 days 192 days	9	San'm 6 yrs.	7 yrs.	Pos. sputum. X-ray—cavity left upper. Hemoptysis-25 x	Improved since 1-18-38. Sputum neg. Ant. stage necessary for remaining 1½ oz. daily sputum
5	B.J.	M	W	28	R	2	30 days	7	San'm 4 yrs.	4 yrs.	Pos. sputum. X-ray—bilateral involv. with cavity rt. upper	Improved since 6-15-37. No sputum. Opposite lung healing
6	S.M.	F	W	23	R	2	90 days	6	Rest 1½ yr.	1½ yr.	Pos. sputum. X-ray—cavity rt. upper	Well since 2-18-38. Neg. sputum
7	F.M.	M	W	25	L	2	60 days	7	San'm 3 yrs.	3 yrs.	Pos. sputum. X-ray—cavity left upper	Improved since 1-10-38. Sputum pos. but reduced. Needs further collapse
8	Y.H.	M	W	34	R	2	90 days	7	San'm 5 yrs.	5 yrs.	Pos. sputum. X-ray—cavity rt. upper	Improved since 1-5-38. Dyspnea after second stage. Pos. sputum—needs further collapse
9	J.Y.	F	W	34	L	2	30 days	6 scap.	San'm 4 yrs.	4 yrs.	Pos. sputum. X-ray—cavity left upper	Improved since 3-9-38. Sputum decreased
10	E.B.	M	W	39	R	1	.....	3	San'm 4 yrs.	4 yrs.	Pos. sputum. X-ray—cavity rt. upper	Improved since 3-9-38. X-ray and sputum improvement but both positive. Contralateral temporary flare-up. Refuses further surgery
11	C.G.H.	M	W	23	R	3	596 days 21 days	7	San'm 3½ yrs. Phren. 1 yr.	3¾ yrs.	Pos. sputum. X-ray—multiple cavities upper rt	Well since 2-27-38. Severe diabetes. Prev. history septic bilateral parotitis, rt. TB empyema. No surgical complication
12	R.J.K.	M	W	34	R	1	.....	3	San'm 2 mo.	4 yrs.	X-ray—honeycomb lesion rt. apex	Well since 12-10-36. Never had sputum
13	E.J.G.	M	W	36	L	1	.....	5	San'm 5½ yrs. Phren. 3 yrs.	6 yrs.	Pos. sputum. X-ray—cavity and honeycomb left apex	Well since 8-6-35. Extensive one-stage operation. High diaphragm from phrenic op.
14	E.P.	M	W	24	R	1	.....	3	San'm 8 mo. Pneum. 8 mo.	8 mo.	Pos. sputum, hemoptysis. X-ray—cavity rt. upper	Improved since 12-4-36. Refused further surgery. Working. No further hemoptysis. Sputum pos.
15	C.C L.	M	W	26	R	4	43 days 665 days 90 days	7	San'm 1½ yrs.	4 yrs.	Pos. sputum, hemoptysis. X-ray—7 cm cavity rt. apex	Well since 1-31-38. Had refused further surgery after first stage until hemoptysis. Anterior stage done

No.	Case	Sex	Race	Age	Side	Stages	Stage Interval	No. Ribs	Prelim. Treatment	Duration of Symptoms	Surgical Indication	Remarks
16	G.H.	M	W	30	L	2	32 days	4	San'm 1½ yrs. Pneum. 16 mo.	2 yrs.	Pos. sputum. X-ray cavity left upper, honeycomb both uppers	Well since 11-30-37. Working. Opposite lung cleared
17	W.T.	M	C	30	L	1	.....	5	San'm 5 yrs. Phren. 3 mo.	6 yrs.	Pos. sputum, hemoptysis. X-ray—cavity and honeycomb upper left	Well since 1-21-36. Streaked sputum for 178 days postoperative. Now asymptomatic and working
18	O.S.	M	C	20	L	2	64 days	5	San'm 8 mo. Phren. 3 mo.	15 mo.	Pos. sputum, chills, fever. X-ray—cavity and honeycomb whole left	Improved since 3-7-36 Refused further collapse. Asymptomatic working
19	J.C.	M	C	30	R	1	.....	3	San'm 5 mo.	13 mo.	Pos. sputum. X-ray—7 cm. cavity rt. upper	Well since 4-16-37. Refused further surgery. Cavity closed; sputum negative
20	E.E.D.	F	W	25	R	3	73 days 50 days	11	San'm 1 yr. Phren. 6 mo.	4 yrs.	Pos. sputum, hemoptysis. X-ray — 6 cm. basal cavity rt	Well since 8-8-36. Moderate kyphoscoliosis; no complaint
21	A.K.	M	C	20	L	2	19 days	11	San'm 9 mo. Phren. 2 mo. Thoracotomy 2 mo.	19 mo.	Pos. sputum, chills, fever. X-ray—multiple cavities to 4 cm. with mixed empyema left	Well since 4-16-37. Syphilis 4 plus, controlled by mapharsen. Open thoracostomy necessary for tension pneumothorax. Oleothorax P.O. for empyema pocket
22	E.V.Mc.	F	W	23	L	3	21 days 35 days	7	San'm 2 yrs. Pneum. 18 mo.	4 yrs.	Pos. sputum. X-ray—4 cm. cavity left upper	Well since 1-22-36. P.O. pulmonary embolus third stage. Recovery
23	Z.L.P.	F	W	31	L	3	49 days 27 days	7	San'm 10 mo. Pneum. 8 mo.	20 mo.	Pos. sputum. X-ray—cavity left upper. Inefficient pneumothorax	Well since 2-27-38. Closed pneumolysis unsuccessful—2 attempts
24	H.M.H.	F	W	25	R	2	36 days	5	San'm 18 mo.	20 mo.	Pos. sputum. X-ray—honeycomb rt. upper	Well since 5-14-37
25	M.K.G.	F	W	25	L	2	85 days	6 with scap.	San'm 3 yrs. Pneum. 4 yrs.	4¾ yrs.	Pos. sputum. X-ray—10 cm. cavity left upper. Honeycomb rt. apex	Improved since 2-17-37. Large cavity closed. Needs bilat. collapse but refuses. Pneumolysis failed
26	M.V.L.	F	W	32	R	2	8 mo.	5	San'm 3 yrs. Phren. 1 yr.	3½ yrs.	Pos. sputum. X-ray—multiple cavities to 4 cm. upper rt.	Well since 3-26-37
27	A.C.L.	F	W	24	R	4	145 days 30 days 30 days	8 with scap.	San'm 5 yrs. Pneum. 2 yrs.	7 yrs.	Pos. sputum. X-ray—12 cm. cavity rt. upper	Well since 3-3-37. First stage ineffective Wilms-Sauerbuch. Anterior stage done. Had P.O. third stage lobar pneumonia. Recovery
28	L.D.	F	W	31	L	2	20 days	11	San'm 10 yrs. Phren.	10 yrs.	Pos. sputum. X-ray—diffuse honeycomb whole left. Hemoptysis	Well since 8-14-35. Extensive 5-rib first stage—graded second stage
29	C.M.M.	F	W	40	R	3	140 days 39 days	5	San'm 11 yrs.	14 yrs.	Pos. sputum. X-ray—honeycomb upper rt. marked fibrosis	Well since 3-30-38. Limited vital capacity (800 c.c.) requiring single rib removals
30	E.M.C.	F	W	34	R	2	53 days	5	San'm 16 mo. Phren. 7 mo.	1½ yrs.	Pos. sputum. X-ray—8 cm. cavity rt. upper	Unfinished. Improved since 3-1-38

* Sanatorium regimen.
† Pneumothorax (artificial) previous to surgery. All cases had trials, mostly failures.
‡ Phrenic interruption on the side operated (temporary).

Transverse processes are removed where the cavitation is large or centrally located. Generally speaking, basal cavities are more difficult to close than apical cavities of comparable size. Resection is not so radical is cases where honeycombing was the sole lesion; every effort is made to leave the patient with as much breathing space as possible. On the other hand, some cases require additional anterior collapse.

We have found ethylene-oxygen-local the anesthetic agent of choice. Patients are urged routinely to rid the trachea of secretions before the induction of anesthesia and again before leaving the operating room. To facilitate this, the operation is carried out in slight Trendelenburg position, and the patient placed in bed for four hours in this position. A small dose of nembutal is given the night before surgery and morphine or pantopon immediately before the operation. No atropine is given as it tends to thicken secretions. Operating time is reduced to a minimum without sacrificing careful technique. Patients are given glucose in saline routinely postoperatively and occasionally acacia is added where the pulse volume is weak. In no instance have we had to give transfusion of blood, but we would recommend it where there has been much blood loss. A soft diet is encouraged by the second or third day and the patient allowed a back rest by the fourth day. As a rule, most patients are out of bed by the eighth day. We have always encouraged our patients to be ambulant at least a week before operation. Oxygen is given if cyanosis or dyspnea develops.

While the series of cases we are reporting is not large, we believe it to be significant of nearly every type of surgical tuberculosis encountered. Moreover, unlike sanatorium cases, most of these patients elected surgery as private patients, and not a few were operated on in general hospitals. In the series there were seventeen male and thirteen female patients; twenty-six were white and four colored. All but one were classified as far advanced cases. All had had previous sanatorium management, the average duration of which was thirty-four months. The average duration of symptoms was 4.2 years; one patient with cavernous tuberculosis had been treated expectantly for fourteen years. The left side was operated on in fourteen patients and the right is sixteen. Eight patients had one stage, fourteen had two, six had three and two had four stages. There was one supplementary anterior thoracoplasty. The average number of ribs removed was 5.7. The average interval between stages was forty-five days; this was computed after discounting instances where the interval was 145, 192, 240, 596 and 665 days in patients who could have been operated sooner but who had to defer surgery because of circumstances beyond their control.

The results of our series were gratifying. There was no mortality; moreover, eighteen patients, or 60 per cent, were cured, ten, or 33 per cent, were improved, and two were unfinished at the time of this report. Of the improved classification we believe that the majority could be made eligible for cure if they elected further surgical collapse. Sooner or later recurrence of symptoms is bound to occur, and we strongly urged such patients to continue treatment with this in mind. One patient had severe diabetes with blood sugar levels before operation as high as 420 mg. per 100 c.c. However, no complications occurred in this case, and the patient subsequently underwent a bilateral cataract operation with like success. It was interesting to note that the insulin requirement in this case was reduced from 150 units daily before operation (when the patient was bed-ridden) to 35 units postoperatively, and this in spite of a gain in weight, increase in diet and a marked increase in energy output. All patients had sufficient vital capacity except one whose measure was 800 c.c. In this case as in another incomplete case, operation was carried out by single rib removals to insure recovery.

There were various sized cavities in this series, the largest being 10 by 12 cm. We have tried to emphasize that any lesion

comprising 25 per cent pulmonary tissue in one lung must have permanent collapse, and it is our feeling that much of the honeycombing that we see is due to re-expansion of inadequately collapsed cases of artificial pneumothorax. Hemoptysis was a common complaint in such cavity cases and it frequently persisted for some time after operation. One patient had had twenty-five severe hemorrhages before coming to surgery. Another was subjected to the old Wilms-Sauerbruch operation for a relatively large cavity; this took three stages of secondary thoracoplasty for eventual closure. Our aim was cavity closure in each instance so that the walls of the cavity would be held closed permanently after fibrosis was established.

There was but one case of postoperative flare-up with recovery. Any sign of exudative inflammation as evidenced by interval *x*-rays was sufficient to postpone further surgery. There was one postoperative pulmonary embolus with recovery. One patient had a postoperative wound infection which we believe came from a crust at the site of a previous scar. In this regard, we emphasize rigid asepsis both before and at operation. Green soap and water, repeated, then ether and mercresin constituted our routine.

CONCLUSIONS

We have not presented anything new in the surgical management of pulmonary tuberculosis. However, we wish to re-emphasize certain conclusions which we believe must be accepted if good results are to be obtained.

1. The type of operation has been well standardized and is relatively safe; there were no deaths in this series of consecutive cases representing sixty-two stages of operation.

2. There are certain requisites for surgical collapse as well as for surgical risk. Contralateral disease is no contraindication; as a matter of fact, the unoperated side seemed to improve with each stage of operation where involvement had been the result of cross infection from the involved side.

3. Minimal sacrifice of pulmonary tissue but sufficient collapse for cavity or honeycomb closure is paramount.

4. The results of this series of cases are encouraging. Although only 60 per cent of our patients are well,* the 33 per cent who were improved are eligible for the "well" category if they can be persuaded to elect further surgical collapse. There were no unimproved patients in the series.

REFERENCES

Alexander, John. Personal communication.

Alexander, John. The Surgery of Pulmonary Tuberculosis. Philadelphia, 1938. F. A. Davis Co.

Behrend, M., et al. Thoracoplasty in tuberculosis of the lungs. *Dis. of Chest*, 3: 8–13 (Sept.) 1937.

Buckingham, W. W. Surgery in pulmonary tuberculosis. *Dis. of Chest*, 2: 17–19, 36 (May) 1936.

Eloesser, L. An operation for tuberculous empyema. *Dis. of Chest*, 1: 8–9, 23 (Oct.) 1935.

Harrington, S. W. Surgical treatment of pulmonary tuberculosis. *Minnesota Med.*, 17: 174 (April) 1934.

Hedblom, C. Results of surgical treatment of pulmonary tuberculosis. *Am. Rev. Tuberc.*, 32: 1–31 (July) 1935.

Lilienthal, H. Pulmonary tuberculosis. Recent types of operation. *J. A. M. A.*, 102; 1197 (April 14) 1934.

Nixon, J. W. Personal communication.

*The term "cure" has been used synonomously with "well." This does not conform to the symbol as used by the National Tuberculosis Association.

EMPYEMA THORACIS

EXPERIENCES IN THE TREATMENT OF 104 CASES EMPLOYING A DOUBLE-CHANNEL CATHETER FOR DRAINAGE

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THE diagnosis of empyema thoracis having been made, it is accepted by many authors that the selection of a method or combination of methods to be used in treatment must be governed by the individual, clinical features and requirements of the case. The division of the profession into two camps, one advocating the so-called "open method" and the other the so-called "closed method," is unfortunate.

Surgically speaking, empyemas have been divided into two groups, the synpneumonic and the postpneumonic types. These conditions, although both bear the label "empyema," are different in every respect. No method of treatment can be intelligently selected without a thorough appreciation of these differences and their significance. The following points of contrast will make clear the most important differences between them:

Synpneumonic Type	Postpneumonic Type
1. Pus accumulates early in the pneumonia, sometimes even before the consolidation is apparent.	1. Pus accumulates late in the pneumonia, or even after the pneumonia has resolved.
2. Cultures yield the streptococcus, or, less frequently, the colon bacillus, alone or as the predominating organism in a mixed pyogenic infection.	2. Cultures yield the pneumococcus alone or as the predominating organism in a mixed pyogenic infection.
3. Onset is not associated with a change of temperature but the pneumonic fever continues as a sustained rise.	3. Onset is marked by a resumption of fever, which assumes septic characteristics.
4. Toxicity is marked.	4. Toxicity is slight.
5. Pus is thin with few fibrin clots.	5. Pus gradually becomes thick with much coagulated fibrin.
6. There is rapid involvement of the general pleural cavity, adhesions forming late.	6. There is a tendency to localization, with early formation of adhesions.

There is no reason to doubt the sincerity or honesty of any of the authors who have reported their individual methods of treatment of this disease. It must, therefore, be deduced that success depends more upon the individual than upon the method which he uses.

The method of choice in drainage depends upon the underlying pathologic physiology which requires correction. Of paramount importance are the following:

1. The subatmospheric pressure normally within the pleural cavity.
2. The infecting organism.
3. The presence or absence of localizing adhesions.
4. The site at which pus most directly contacts the chest wall.
5. The amount of fibrin clots present.
6. The patient's general clinical condition.
7. The presence or absence of bacteremia.

Other things being equal, a lung upon the autopsy table responds to the same physical laws as a lung within the pleural cavity. In other words, a consolidated lung will sink and an aerated lung will float. Except, therefore, in massive empyemas, involving the whole pleural cavity, or those which form in the presence of important preëxisting adhesions, the re-aerated lung in postpneumonic empyema will float and the greater amount of pus will be found in the posterior costal gutter. The consolidated lung in synpneumonic empyemas, on the other hand, will sink and the pus will be displaced upward to the anterior chest wall. Exception, perhaps, should be made for those cases of streptococcus empyema, which occur in the absence of any demonstrable consolidation. It

becomes immediately apparent that the physical and *x*-ray signs must be interpreted in the light of this simple fact. The distribution of the pus may also differ according to the position occupied by the patient in bed during the sojourn of the pneumonic consolidation. In one of the author's cases, bilateral empyema developed while the patient was in a respirator. The longitudinal distribution of the pus in the pleural cavity was so uniform that the *x*-ray failed to give diagnostic evidence of its presence.

It is also apparent that the routine choice of the eighth interspace in the midscapular line as the site of election for diagnostic puncture or for drainage cannot be expected to yield the best results.

In the average case, the diagnosis of empyema is easy, as is also the localization of pus by diagnostic tap. It is clear, however, from the remarks of the preceding paragraph, that mistakes can readily be made. If the pus is loculated, it may be apically placed, supradiaphragmatic or interlobar; or there may be several pockets either isolated or communicating. In such instances, the same care must be taken in localization as would be given a lung abscess.

In general, it may be said that, if any difficulty is encountered in obtaining pus at thoracentesis, or in demonstrating an ample amount of free space with the aspirating needle, *x*-ray localization is necessary. This can generally be accomplished by taking, in addition to the usual postero-anterior film, an anterior and posterior oblique view of the affected side, especially after the replacement of some pus by air. It goes without saying, of course, that, in multilocular accumulations, the greatest care must be taken and drainage effected at more than one point if necessary.

In patients who remain immobile over long periods of time in bed, the cellular elements in the pus settle to the bottom of the pleural cavity. It is then possible to obtain by aspiration an entirely erroneous idea of the percentage of pus in the effusion, depending upon whether the heavy sediment or the relatively clear supernatant liquid is drawn out.

The cure of an empyema takes place by the obliteration of the pleural cavity through synechia of the opposing surfaces. No empyema can be looked upon as cured until synechia is complete, and the faster this fusion can be induced, the more quickly is a cure obtained.

In open drainage the process of healing is a progressive adhesion of the two surfaces, beginning at the periphery of the empyema pocket and moving inward. Clean exudative pleural surfaces, when brought into contact with each other, will fuse. It is apparent, therefore, that fusion will take place only when there is no pyogenic membrane and when the production of exudate has ceased, i.e. when the underlying inflammatory process has subsided. It is logical to assume that when these conditions exist, the progress of healing is accelerated by approximating the surfaces of the pleura. The application of negative pressure to the pleural cavity not only provides mechanically for this approximation, but in so doing causes the removal of the pyogenic membrane by friction of the two surfaces. In acute cases, the author has seen this process completed in the remarkably short time of ten days.

The choice of time and method in drainage is much facilitated by this knowledge. Unless dyspnea, toxicity, or remote complications, such as pyelitis or nephritis, demand early interference, nothing is to be gained by drainage before the pneumonic process is cleared up. The patient suffers less inconvenience if he is relieved symptomatically until such time has arrived by repeated aspiration. It is not always easy to judge the condition of the underlying lung, and, in case of doubt, early drainage under strictly closed precautions is better than allowing the defensive thickening of the pleura to proceed too far, inviting the complication of bronchial fistula or chest wall abscess.

Below is presented a method of drainage, which I have used with considerable success. I have no desire to urge its general adoption. On the contrary, I believe that when the fundamental principles are observed, an individualized technique is more likely to assure the attention to detail which is so essential to success. There is a persistent opinion among a certain group of men, however, that treatment cannot be begun until a rib has been resected. This attitude is dangerous and reactionary. Even its advocates admit its disadvantages in children, where the problems faced are the same as in adults, but where the risks of anesthesia, open pneumothorax and mediastinal flutter are naturally increased. Rib resection still has its place in the treatment of empyema but, where proper nursing and supervisory care are available, complete drainage under all circumstances in the acute cases is possible without its use. If the operator proceeds without prejudice upon the assumption that empyema per se should never be a cause of death, it is generally easy to decide upon the right thing to do at the right time.

It is unnecessary to enter into the general physiologic and anatomic principles governing the treatment of empyema, since the subject has been treated comprehensively in a vast literature already extant. Studies made by Graham,[1] Connors,[2] Fitzgerald,[3] Kenyon,[4] Holt,[5] Harloe,[6] are especially recommended for the perusal of the reader who wishes to go more deeply into the general aspects of the subject.

TREATMENT OF CHRONIC EMPYEMA

By chronic empyema is meant, in this group of cases, patients who have been draining pus externally or internally from the pleural cavity for two months or more without showing signs of improvement.

The causes of chronic suppuration in empyema are: (1) foreign body in the pleural cavity; (2) osteomyelitis of the ribs; (3) specific infections; (4) bronchopleural fistula; (5) inadequate drainage; and (6) pleural dead space. This paper concerns itself chiefly with the application of negative pressure to the treatment of empyema. It is obvious, therefore, that a discussion of the above enumerated problems goes beyond its intended scope. They shall be considered only as they are related to the author's method of treatment.

1. *Foreign body in the pleural cavity* should always be ruled out before the treatment of chronic empyema is begun. When present, it can be removed by pleuroscopy without operation and without anesthesia, local or general. Irrigations under negative pressure with the catheter inserted through the sinus hastens recovery.

2. *Osteomyelitis of the ribs* was the only associated condition requiring rib resection in the seventeen cases of chronic empyema reported herewith. In two cases, it was the only condition present, the empyema having been satisfactorily treated in one case fifteen years previously.

3. Of *specific infections*, the commonest offender is the tubercle bacillus. Negative pressure irrigations can be successfully used to keep the pleura clean, to bring about expansion of the lung and to prevent amyloidosis. The underlying tuberculous pulmonary lesion can be treated by staged thoracoplasty at the same time. Experience to date would indicate that synechia of the pleural surfaces is not likely to take place. Some reduction, however, in the volume of pleural dead space may be obtained, and the radical nature of the Schede operation as at present used may be limited in direct proportion to the success of efforts in this direction.

4. *Bronchopleural fistula* is a much more frequent complication of empyema than is commonly believed. In a series of twenty-five consecutive cases, the writer injected methylene blue into the pleural cavity at the time of the initial thoracentesis. It was expectorated in ten cases (40 per cent). This is undoubtedly a higher average than would be found in a larger series of patients, but it emphasizes the fact, never-

theless, that bronchopleural fistula should always be kept in mind as a possibility. In its presence, negative pressure cannot be used and irrigations must be used with great caution, if at all, on account of the danger of flooding the lung. It should not be looked upon as a serious problem in patients not previously operated on. In this series, spontaneous closure took place in all cases in from five to seven days following the initiation of drainage.

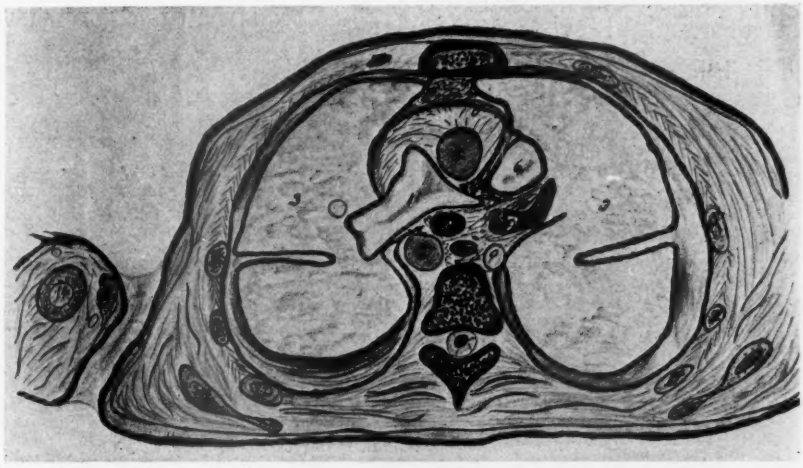

Fig. 1. Cross section of human thorax just below the bifurcation. The close relation of the pleural cavity to the posterior bronchial wall is clearly shown, especially on the right. The relation is equally close on the left, but slightly lower. The bronchi are indicated at 7.

If a cross section of the human body is made at the level of the bifurcation, the parietal pleura is found to be in close relation to the posterior wall of the stem bronchus on both sides. (Fig. 1.) The bronchi are deficient in cartilage posteriorly and have no external protection except a thin layer of loose areolar tissue. They are, therefore, directly subjected to the effects of pus confined within the pleural cavity under increased pressure. Here, then, is the easiest and most direct route for perforation into the airways. I have been able to establish this as the site of fistulization in four cases; two by bronchoscopy, one at autopsy and one by lipiodol radiography. (Fig. 2.) The anatomic location explains the frequency and rapidity with which these openings close, since, as soon as drainage is established, they are occluded by the re-expanding lung as it gradually occupies the posterior costal gutter.

5. *Inadequate drainage* as a cause of chronic empyema results from: (a) dependence upon an opening which is too small to evacuate the pleural cavity; (b) allowing the external fistula to close before the lung is fully re-expanded; or (c) improperly selected drainage site. The first two are faults frequently encountered in closed drainage cases. They may be corrected by rib resection if this method is used early enough to prevent extreme thickening of the pleura. A cure can be obtained more quickly, however, and without rib

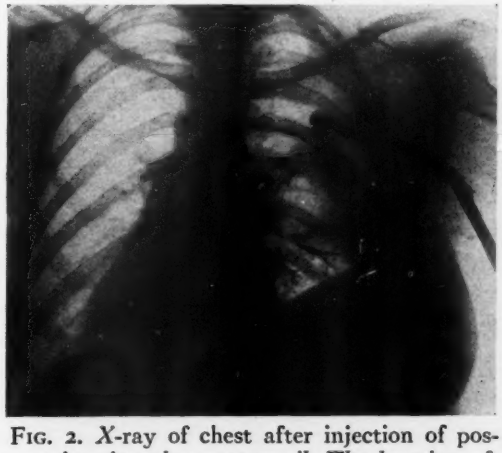

Fig. 2. *X*-ray of chest after injection of posterior sinus by opaque oil. The location of the fistula in the right bronchus is clearly indicated. (See Figure 1.)

resection, by the method to be described later. In the third instance—improperly selected drainage site—the fault lies in improper localization of the pus. The tube may be inserted too high, leaving a collection of stagnant pus below it. It may be too near the periphery of a localized pus pocket. The opening will then be quickly obstructed by the rising diaphragm or the re-expanding lung.

6. In the treatment of chronic empyema with *pleural "dead space"* as the only apparent cause of chronicity, the application of the method to be described finds its greatest value.

It has been claimed by some authors that, when the lung fails to re-expand, an atelectasis, due to bronchial obstruction, should be suspected. I have not been able to find in my personal experience or in bronchoscopic examinations in the services of other men, any clinical evidence in support of this theory. On the contrary, even if obstruction of the bronchus were found, it would have to be looked upon as the effect, rather than the cause, of the thickened pleura and the collapsed lung.

The removal of the obstruction should, therefore, be sought as a natural result of the restoration of function by re-expansion of the lung. In seventeen chronic cases (see summary Table II) the empyema was cured without rib resection; in eleven cases this was accomplished by causing a partially atelectatic lung to re-expand. The single or combined effect of removal of foreign body from the pleural cavity; the correction of the drainage site, and the application of negative pressure, either through the old, or through the corrected site of drainage brought about the result. In seven of these cases, I am convinced, a cure without operation must be attributed to the beneficial effect of negative pressure on a clean and gradually thinning pleura. At least, it has been demonstrated beyond doubt that, in the absence of osteomyelitis of the ribs and specific infections of the pleura, no major surgical procedure should be embarked upon without giving the method a fair trial. It has never failed in my cases except in the presence of the two conditions mentioned above.

METHOD OF DRAINAGE

This method was first described in 1933. Up to that time, no record could be found in the literature of the use of a two-way catheter in the treatment of empyema. Recently, however, Bloxsom[7] has described a set-up utilizing a return-flow glass douche bulb by which the same principles of treatment may be applied.

In the author's method,[8] a flat catheter, 13 mm. wide, with two size 18 French channels, is inserted intercostally by a specially-designed trocar and cannula. (Fig. 3.) If there is no bronchial fistula, the branches are connected to a bottle containing normal saline solution on one side of a supporting standard and to a syphon system on the other side. (Fig. 4.)

It will immediately be seen that variations in the application of pressure to the pleural cavity may be regulated by changing the levels of the saline bottle or of the drainage bucket. In practice, however, it has been found best to initiate drainage with the relative levels as shown. Inflow and outflow may be regulated by pressure of the fingers on the corresponding tubes. The content of the pleural cavity is alternately increased and decreased, using hot salt solution. In this way, the collapsed lung is gently "massaged" until evacuation of the pus is complete, a period which need not occupy more than thirty minutes. Continuous irrigation under constant negative pressure is then started. In order to avoid too great an exertion of negative pressure in the pleural cavity, the drainage bucket is gradually raised. The best operating position has been found to be wheer the water level in the drainage bucket is from 12 to 16 inches below the water level in the syphon bottle when this level is in the same horizontal plane as the intercostal tube.

In the presence of bronchial fistula, it is evident that neither negative pressure

nor the free use of irrigating solutions can be used. The apparatus is then set up as shown in Figure 5. Bronchial fistula in the ordinary post-pneumonic empyema, in the opinion of the writer, closes more quickly if the development of an intrapleural positive pressure is prevented. This is accomplished by attaching the outflow side of the catheter to the ordinary "water seal." It must be noted, however, that this device is not effective unless there is (*a*) direct and continuous downhill flow from the chest wall to the drainage bottle and (*b*) constant maintenance of the outlet of the drainage tube at a level not more than 1 cm. below the surface of the water forming the seal. It is, therefore, necessary, to avoid loops, kinks and pendancies in the outflow tube. The inflow tube may be used with a Murphy drip, with intermittent irrigation of suitable quantity, or without irrigation, as the case requires. Most fistulas will close in from five to seven days. The arrangement of bottles is then changed to permit of negative pressure irrigations as in Figure 4.

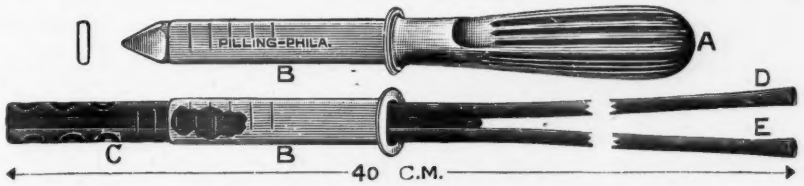

Fig. 3. Special trochar, cannula and two-way catheter. (Modified from the original of Dr. Norman Bethune.)

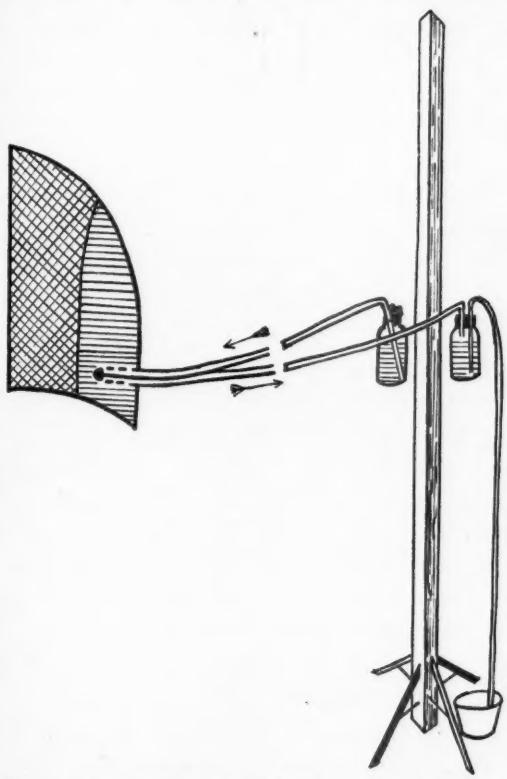

Fig. 4. System for the initiation of drainage in the absence of bronchial fistula.

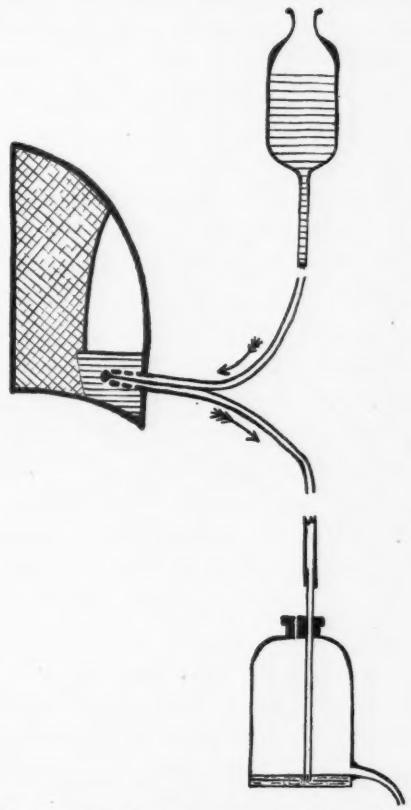

Fig. 5. Drainage system in bronchial fistula. (See text.)

The maintenance of an airtight contact is provided by the use of a rubber sponge dressing. An ordinary bathroom sponge of fine texture, not less than 1 inch in thickness, is sterilized. After the insertion

of the catheter, the sponge is perforated by a pointed clamp without tearing. The catheter is seized and drawn through. The sponge is applied directly to the chest wall and strapped down by adhesive tape.

The presence of large masses of coagulated fibrin, found especially in pneumococcic empyemas will interfere with drainage. This difficulty, however, is easily overcome. If coagulum is known to be present in the pleural cavity, it may be evacuated when drainage is established, in many instances. The caliber of the author's special cannula is large enough to permit its escape, except where particularly large quantities are present, or where it is considered unwise to empty the pleural cavity quickly. In such instances, the catheter is inserted and the pus removed in the manner described below.

During the first three to four days, drainage is maintained by alternating the direction of flow in the branches of the catheter. This allows time for the coagulum to detach itself from the pleural surfaces. The catheter is then removed; the patient is tilted to the affected side and asked to cough. As the clots are forced out of the sinus, they are seized by forceps or a hemostat and withdrawn. In this way, I have removed as much as a pint of coagulum in large masses, without technical difficulty and without embarrassment to the patient. Following this, the catheter is reinserted and irrigations may be continued without interruption.

At the end of the first two days of drainage, the return from the pleural cavity is clear. Since the pleural cavity is empty, toxic absorption does not take place. The inflammatory membrane covering the pleural surfaces is dissected free by the action of the digestive ferments of the underlying white blood cells. With the lung maintained in a state of re-expansion by an appropriate degree of negative pressure, friction of the pleural surfaces rapidly removes this membrane. It can be seen to pass out of the drainage tube in the form of whitish flocculent masses. Actual pathologic section of a twenty-four hour accumulation of this material shows that it consists of partially digested, fibrous and granulation tissue, with entangled, amorphous débris. This action is particularly important in the case of chronic empyemas where a thickened and inelastic pleura may be the sole barrier to the obliteration of pleural dead space.

As soon as this sloughing-off process is complete, two clean exudative surfaces find themselves in intimate contact and fusion rapidly takes place. The tube is removed when the volume of the pleural pocket is not more than 25 to 35 c.c. A section of rubber dam is inserted to maintain patency of the sinus until the space is completely obliterated. The closure of

TABLE I
ETIOLOGY IN 104 CASES

	No. of Cases	No. of Deaths	Per Cent Mortality
I. Not post-pneumonic:			
Complications of			
Thoracic operations	3	1	
Abdominal operations	5	1	
Subdiaphragmic abscess	1	1	
Trauma to chest	1	1	
Total	10	4	40
II. Post-pneumonic:			
Acute			
Under 5 years	13	1	7.7
Over 5 years			
Unilateral	60	4	6.25
Bilateral	4	0	
Chronic	17	0	
Total	94	5	5.31

TABLE II
CHRONIC EMPYEMA

Cause of Chronicity	
Foreign body in pleural cavity	2
Osteomyelitis of ribs	4
Inadequate drainage	6
Incomplete expansion of lung (pleural thickening)	5
Total	17

No case with tuberculous, or other specific infecting agent is included.

this residual space often requires as long, or even longer, than the adhesion of the entire pleural surfaces when they are kept constantly clean and continuously applied to each other.

SUMMARY OF CASES

Elapsed time since the initial drainage, in these cases varied from three months to six years, the average duration being one year, approximately. In the case of six years' duration, the patient still has a residual sinus from infected bone but has been able to resume his normal activities.

COMMENTS

Of the acute cases treated by drainage, eighteen were in patients over five years of age, unilateral and uncomplicated. These cases were cured in an average of 14.6 days.

One patient was cured by two aspirations of an empyema yielding a pure culture of Staphylococcus aureus.

In one case of bilateral empyema yielding a pure culture of hemolytic streptococcus, the right side was drained. The left side cleared up after a single aspiration.

Three patients, in whom a late diagnosis was made, recovered after rupture into the trachea, without further drainage.

Causes of death in the five cases reported in post-pneumonic empyemas were as follows: (1) intractable diarrhea in a 7 months old infant (the empyema wound was closed two weeks before death); (2) embolus to the brain (?); (3) hemolytic streptococcus septicemia; (4) pneumonia in the contralateral lung (two cases).

Sulfanilamide and sulfapyridine were used by various physicians in adequate dosage, in sixteen cases, with the following results:

1. Drugs administered after the diagnosis of empyema and after the apparent resolution of the pneumonia—six cases—no effect.

2. Drug administered during the pneumonia and before the diagnosis of empyema: three patients had sterile effusion (exudate) at first aspiration; one patient became sterile one week after first aspiration, at which time a pure culture of anaerobic streptococcus had been obtained; three patients became sterile after two weeks drainage and needed no further treatment; three patients had clinically and *x*-ray diagnosed effusion which disappeared spontaneously.

It would appear therefore that when these drugs (especially sulfapyridine) are administered in pneumonia, the complicating effusion, if any, is likely (1) to be sterile throughout, or (2) to become sterile under treatment more rapidly than would be the case in the absence of the drug.

SUMMARY AND CONCLUSIONS

1. A method of treating empyema is described, which permits of the regulated application of negative pressure to, and constant irrigation of, the pleural cavity.

2. The apparatus is extremely simple and easy to operate. Rib resection is unnecessary, even in the presence of large amounts of coagulum, but adequate nursing and supervisory care are essential.

3. Ninety-four cases of post-pneumonic empyema are reported with a death-rate of 5.31 per cent. No deaths were due to the empyema.

4. Uncomplicated post-pneumonic empyemas should be cured in from two to three weeks.

5. Observations are made from the author's experience in connection with the physiology and anatomy of open and closed pneumothorax and bronchopleural fistula.

6. Evidence is presented in support of the author's belief that the obliteration of the pleural cavity is accelerated by the use of negative pressure and the maintenance of two clean granulating surfaces in apposition.

7. The results of the application of the same principles in the treatment of chronic empyema are shown. Seventeen cases were

treated, with no deaths and without rib resection except for osteomyelitis.

REFERENCES

1a. Graham and Berck. Principles vs. details in the treatment of acute empyema. *Ann. Surg.*, 98: 520 (Oct.) 1933.

1b. Graham, E. A., and Bell, R. D. Open pneumothorax. Its relation to the treatment of acute empyema. *Am. J. Med.*, 156: 839, 1918.

2. Connors, J. F. The treatment of empyema. *Ann. Surg.*, 96: 1092, 1934.

3. Fitzgerald, R. R. Acute empyema in infants and children. *Canad. M. A. J.*, 31: 479–485, 1934.

4. Kenyon, J. H. A preliminary report of a method of treatment of empyema in young children. *M. Rec.*, Oct. 21, 1911.

5. Holt, L. E. The siphon treatment of empyema in infants and young children compared with other measures. *Am. Med.*, 8: 381, 1913.

6. Harloe, R. E. Empyema. *Am. J. Surg.*, 26: 231–247, 1934.

7. Bloxsom, A. P. Empyema: a method of treatment with continuous irrigation and drainage. *J. Thor. Surg.*, p. 698, Aug., 1937.

8. Lloyd, M. S. A new application of the principle of suction irrigation in the treatment of empyema thoracis. *J. Thor. Surg.*, 3: 538, 1934.

9. Empyema Commission. Cases of empyema at Camp Lee, Va. *J. A. M. A.*, 71: 366–442, 1918.

Venous thromboses may be associated with, if not actually a reflex cause of, arterial spasm serious enough in some cases to induce gangrene of the limb.

From—"Circulatory Diseases of the Extremities" by John Homans (Macmillan).

THE TREATMENT OF VENEREAL LYMPHOGRANULOMA WITH SULFANILAMIDE*†

A PRELIMINARY REPORT

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THE purpose of this communication is to present evidence to show the effectiveness of sulfanilamide in the treatment of the anorectal and inguinal manifestations of venereal lymphogranuloma. The value of sulfanilamide in this disease has been reported by others.[1,2,3,4,5] Shropshear[3] and Knight and David[5] have stressed the value of this drug in the treatment of the anorectal phase of venereal lymphogranuloma, with which we are particularly concerned. However, it should be noted that a precise plan of treatment with sulfanilamide has yet to be evolved.

It is likely that sulfanilamide exerts a specific effect on the virus of venereal lymphogranuloma, and, at least in part, on the secondary contaminants. Our experience supports these possibilities. Whether the beneficial effect of sulfanilamide is temporary or permanent has not yet been established. Observations over a period of time will determine this point.

Our studies appear to show that large amounts of sulfanilamide should be given during the initial period of treatment. We have found that a minimum of 24 Gm. of sulfanilamide should be administered over a period of four days. This can best be accomplished in a hospital, as it is well known that ambulatory patients cannot take large amounts of the drug with safety. Furthermore, it is dangerous to entrust potent drugs to these patients without proper daily medical supervision. Early toxic symptoms may go unrecognized, as they are unpredictable.

After the initial period of treatment in the hospital, our patients were given approximately 3 Gm. of sulfanilamide daily for about three months, preferably without a rest period. We are not yet satisfied that this is sufficient treatment. Another point about which we are in doubt is whether the rectal route of administration of sulfanilamide is superior to the oral in these cases. Our experience with the first case herein reported suggests the former possibility.

During the period of treatment in the hospital, daily determinations of the concentration of sulfanilamide in the blood were made. Sole reliance should not be placed on the amounts of the drug administered because the factors of absorption and excretion vary in each patient. It appears that a concentration of 5 or more mg. of free sulfanilamide per 100 c.c. of blood is essential. In three ambulatory clinic patients, the results obtained with sulfanilamide were poorer than in the patients treated in the hospital. In one of the ambulatory patients, little or no change in the status of the rectal stricture was noted after the daily administration of approximately 3.5 Gm. of sulfanilamide until 50 Gm. were supposed to have been taken. At the end of that time the concentration of sulfanilamide in the blood was 1 mg. free sulfanilamide per 100 c.c. It is difficult to determine how accurately dispensary patients followed our instructions as to dosage, limitation of fluid, etc. This furthers our conviction that these patients do better when treated in a hospital under strict control and supervision.

Sulfanilamide exerted the most pronounced effect on the inflammatory and

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† Sulfanilamide was furnished by the Medical Research Department, Winthrop Chemical Company.

suppurative processes. The scar tissues became more elastic. The perirectal infiltration and exudate disappeared. The raw surfaces healed. The resistance to the examining finger diminished, and the strictures were easily dilatable. In addition to this favorable effect on the strictures, pronounced beneficial changes in the patients' general health were noted.

During the time of sulfanilamide therapy, our afebrile patients were allowed out of bed. Fluids were limited to 2,000 c.c. during twenty-four hours. Four Gm. of sodium bicarbonate in four divided doses were given daily. A regular diet was allowed, except during the period of the rectal administration of sulfanilamide, when a bland, non-residue diet was given.[6] The concentrations of sulfanilamide were determined by the method of Marshall, as modified by Marshall and Litchfield.[7]

Case 1. J. R., a 25 year old negress, was first seen on December 8, 1938, in the Out-Patient Department of the Brooklyn Hospital, because of incontinence of feces, purulent rectal discharge, diarrhea, and dull pain about the rectum. These symptoms had been present since 1935, when she had had several rectal operations at a city hospital. During the previous year she had lost about 40 pounds in weight. Physical examination revealed poor nutrition, evidence of loss of weight, pale mucous membranes, scarring in the anal and perianal regions, superficial fissures, a tight annular rectal stricture situated 4 cm. from the anus, perianal, perirectal and pericolonic infiltration, and purulent discharge. The stricture was about 0.5 cm. in diameter, and could be stretched only with difficulty. The urinalysis was normal. Examination of the blood revealed secondary anemia. (Table 1.) The blood Wassermann test was negative. The Frei test, using two human antigens, was strongly positive. The sedimentation time of the erythrocytes was twenty-five minutes.

Table 1
Fluctuations in hemoglobin (case 1)

Date	Hemoglobin	Erythrocytes	Leucocytes	Polys.	Lymph.	Eos.
1/17/39	50	3,200,000	11,100	67	33	
1/21	..	.....	7,250	61	37	2
1/23	54	3,150,000				
1/24	56	3,350,000	9,950	74	24	2
1/27	57	.....	9,200	67	32	1
1/28	58	.....	6,700	69	28	3
1/29	58	3,210,000	11,850	62	38	
1/30	64	.....	6,650	50	48	2
1/31	64	.....	8,400	62	36	2
2/1	60	.....	10,950	74	26	
2/2	64	.....	6,300	55	43	2
2/3	62	.....	5,400	56	43	1
2/4	60	.....	13,000	73	27	
2/5	54	.....	6,200	68	32	

At various intervals of time, attempts were made to dilate the stricture with numbers 3 and 5 F. Wales rubber bougies, as well as with the index finger. At times, chilly sensations followed these dilatations.

On January 17, 1939, this patient was admitted to the Brooklyn Hospital because of fever ranging from 101 to 103°F. Colonic irrigations were given daily for four days because of the profuse rectal discharge. On January 21, a course of oral neoprontosil was begun, giving 0.6 Gm. per dose five times daily. Twenty-four hours later, the temperature dropped to normal. On January 27, the administration of sulfanilamide per rectum was begun after the preliminary determinations of the blood showed an absence of sulfanilamide. One hundred c.c. of 1 per cent of sulfanilamide solution were administered every four hours, day and night. At times parts of the medication were expelled. After the administration of 6 Gm. of sulfanilamide, the concentration of the blood was 4.5 mg. free and 2.5 mg. conjugated sulfanilamide per 100 c.c. After 12 Gm. had been given, 3.5 mg. free and 2.5 mg. conjugated sulfanilamide were found in each 100 c.c. of blood, and after the administration of 18 Gm. of sulfanilamide, the concentration of the blood was 6.5 mg. free and 1.5 mg. conjugated sulfanilamide per 100 c.c. For the ensuing three days, most of the medication was expelled.

At this time, examination revealed that the index finger was easily inserted through the stricture. On February 2, it was noted that the perianal and perirectal infiltration was disappearing and that the stricture had been favorably modified since admission to the Out-Patient Department. The rectal discharge and the diarrhea ceased after the rectal administration of 5 Gm. of sulfanilamide. The hemoglobin value increased gradually as shown in Table 1.

On February 2, sulfanilamide therapy by mouth was begun, giving 100 c.c. of 1 per cent solution every four hours, day and night. After the administration of 6 Gm., the concentration of the blood was 7 mg. free and 3 mg. conjugated sulfanilamide, and after a total of 12 Gm. of sulfanilamide had been given, 9 mg. free and 2 mg. conjugated sulfanilamide were found in each 100 c.c. of blood.

During the period of oral administration of sulfanilamide, the diarrhea returned, and continued for three days. The hemoglobin value gradually dropped. (Table 1.) However, the status of the anorectal disease continued to show improvement. Almost all of the perirectal and pericolonic infiltration disappeared. The index finger could easily be introduced through the lumen of the stricture. The tissues were softer and more elastic. No pain was produced on rectal examination.

The patient was sent home on February 7, 1939, and was followed up in the Out-Patient Department. Oral sulfanilamide therapy, about 3 Gm. daily, was continued. On March 4, a sigmoidoscopy was performed. A regulation ¾ inch sigmoidoscope was inserted with ease and caused no trauma. The mucous membrane was pale above the stricture. The site of the stricture was reddened. There was no infiltration palpable.

Comment. The striking response to sulfanilamide therapy in this case was remarkable. This response was far superior to any other therapeutic agent employed in our clinic in the past. It should also be noted that the hemoglobin values increased during the time sulfanilamide was administered rectally. When the same amounts of solution of the drug were given orally, the hemoglobin values began to decline. Also, during this time, there was a return of the diarrhea which continued for several days. These observations are worth noting, although at the present time we have no explanation for their occurrence.

Case II. L. P., a 28 year old married negress, was seen in the Out-Patient Department on December 20, 1938, because of obstinate constipation, rectal tenesmus, intermittent rectal bleeding associated with dull pain, and the passage of narrow calibered stool. She had had syphilis two years before, for which she had been treated extensively. Physical examination was essentially normal except for a soft annular rectal stricture, 5 cm. above the anus. There was moderate perirectal infiltration. The infant sigmoidoscope could not be introduced through the stricture. One week later, during an attempt to dilate the stricture with the index finger, a tear to the left occurred which was followed by bleeding. The stricture was thereafter dilated with number 5 F. Wales rubber bougie on several occasions.

A Gram stain of the purulent rectal material showed Gram-negative organisms, and B. welchii. No ova, parasites, amebae or tubercle bacilli were found. The blood Wassermann test was negative. The urinalysis was normal. The Frei test, using two human antigens, was positive. The sedimentation time of the erythrocytes was forty minutes. Cervical smear was negative for gonorrhea.

During the course of her illness, the patient was taken with acute abdominal pain, and on February 11, 1939, she was admitted to the Gynecologic Service of the Brooklyn Hospital with a diagnosis of acute salpingitis. The aperture of the rectal stricture at that time barely admitted the tip of the index finger. Under conservative treatment, including sulfanilamide tablets by mouth, her pelvic inflammatory disease gradually subsided.

On February 19, sulfanilamide therapy, using 1 per cent solution administered rectally, was begun. One hundred c.c. of this solution was given every four hours, day and night; the first midnight dose was omitted. Through an oversight, the preliminary determination of sulfanilamide in the blood was not done. After the administration of 6 Gm., the concentration of the blood was 5 mg. free and 4 mg. conjugated sulfanilamide, and after a total of 12 Gm. had been given, the concentration of the blood was 12 mg. free and 1.5 mg. conjugated sulfanilamide per 100 c.c. A temperature of 102.4°F. with a severe chill occurred. Sulfanilamide therapy was discontinued. The temperature remained at 102.4°F. the next day, and then dropped to normal.

On February 26, the patient was transferred from the gynecologic to the proctologic service. The gynecologists, in the transfer note, stated that the index finger passed the strictured area easily and painlessly.

On February 27, the rectal administration of sulfanilamide was resumed. Preliminary blood

tests showed no trace of sulfanilamide. After the administration of 7 Gm. of sulfanilamide, the concentration of the blood was 6 mg. free and 2 mg. conjugated sulfanilamide per 100 c.c.; after 12 Gm. (midnight dose being omitted), there were 6 mg. free and 1.5 mg. conjugated sulfanilamide; and after a total of 19 Gm. of sulfanilamide had been given, the concentration of the blood was 9 mg. free, and 2 mg. conjugated sulfanilamide per 100 c.c. No reaction was noted.

The patient was sent home on March 9, and has been followed up in the Out-Patient Department.

On several occasions Wales' rubber bougies, sizes 6 and 7 F., were easily introduced into the strictured areas.

Comment. This patient had a stricture with perirectal infiltration. The response to sulfanilamide was as good as in the first case.

Case III. H. H., a 20 year old negro, was seen in the Out-Patient Department of the Brooklyn Hospital on February 1, 1939, because of large, tender inguinal, epitrochlear and posterior cervical glands. The left inguinal glands were considerably larger than those on the right side. Within three weeks, there was considerable swelling and central softening in the left inguinal region, and marked lymphadenopathy on the right side. He was otherwise normal. The past history included gonorrhea (?) which occurred five months previously and was self-treated. The urinalysis was normal. The blood Wassermann test was negative. The Frei test, using three human antigens, was strongly positive.

This patient was admitted on March 2, 1939. The bubo was aspirated, evacuating 20 c.c. of thick pus. Two days later the bubo was found distended, and tenderness continued in spite of a moderate escape of sanguinous fluid from the site of the needle puncture.

On March 9, a course of sulfanilamide therapy was begun. Two suppositories, each containing 0.5 Gm. of sulfanilamide, were given every four hours, day and night. After the administration of 7 Gm., the concentration of the blood was 1 mg. free and 1 mg. conjugated sulfanilamide per 100 c.c.; after 13 Gm. had been given, 2.5 mg. free and no conjugated sulfanilamide were noted (parts of the sulfanilamide were voluntarily expelled on two occasions); after the administration of 19 Gm. of sulfanilamide, the concentration of the blood was 4 mg. free and 2 mg. conjugated sulfanilamide per 100 c.c.; and after a total of 25 Gm. had been given, 6 mg. free and 2 mg. conjugated sulfanilamide were noted for each 100 c.c. of blood.

Following the administration of 13 Gm. of sulfanilamide, the residual suppuration of the left bubo was subsiding, and the pain disappeared. The right lymphadenopathy regressed considerably after the administration of 19 Gm. of sulfanilamide.

Similar courses of sulfanilamide therapy were repeated, giving the same amount of the drug in 1 per cent solution first by rectum, and later by mouth. A total of 75 Gm. of sulfanilamide had been administered.

Comment. The third case is included to show that the inguinal (glandular) type of venereal lymphogranuloma also responds to sulfanilamide therapy. When the concentrations of the blood reached approximately 3 mg. of sulfanilamide per 100 c.c., the pain ceased and the residual bubo began to disappear. The inguinal glands began to regress when the concentration of the blood reached 6 mg. of combined sulfanilamide per 100 c.c. The action of sulfanilamide in this case suggests a specific effect because no secondary infection was present.

SUMMARY AND CONCLUSION

1. Sulfanilamide is very effective in the treatment of anorectocolonic and inguinal manifestations of venereal lymphogranuloma. Its action is most pronounced on the inflammatory and suppurative phases of this disease.

2. Intensive sulfanilamide therapy during the initial period of treatment is advised.

3. The concentration of sulfanilamide of the blood should be determined frequently during sulfanilamide therapy. We arbitrarily advocate a concentration of 5 or more mg. of free sulfanilamide per 100 c.c. of blood.

4. For reasons of safety and effectiveness, sulfanilamide therapy should prefer-

ably be given in a hospital. Daily medical supervision is essential for ambulatory patients.

REFERENCES

1. Shaffer, L. W., and Arnold, E. Lymphogranuloma venereum. *Arch. Dermat. & Syph.*, 38: 705–712 (Nov.) 1938.
2. Torpin, R., Greenblatt, R. B., Pund, E. R., and Sanderson, E. S. Lymphogranuloma venereum in the female. *Am. J. Surg.*, 43: 688–694 (March) 1939.
3. Shropshear, G. Sulfanilamide in the treatment of strictures of the rectum caused by lymphogranuloma venereum. *Illinois M. J.*, 74: 153–156 (Aug.) 1938.
4. Hamilton, G. R. Treatment of venereal lymphogranuloma with sulfanilamide. *Mil. Surgeon*, 83: 431–438 (Nov.) 1938.
5. Knight, A. A., and David, V. C. The treatment of venereal lymphogranuloma with sulfanilamide. *J. A. M. A.*, 112: 527–529 (Feb. 11) 1939.
6. Turell, R., Marino, A. W. M., and Nerb, L. Observations concerning the absorption of sulfanilamide from the large intestine in man. *Brooklyn Hosp. J.*, 1: 90–93 (April) 1939.
7. Marshall, E. K., Jr., and Litchfield, J. T., Jr. The determination of sulfanilamide. *Science*, 88: 85–86 (July 22) 1938.

Varicosity of veins is perhaps most often due to increased intra-abdominal tension such as is caused by heavy labor.
From—"Circulatory Diseases of the Extremities" by John Homans (Macmillan).

A COMPARATIVE STUDY OF SUBTOTAL AND TOTAL HYSTERECTOMIES*

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THE relative merits of total and subtotal hysterectomy have long been a matter of controversy. It is obviously difficult to evaluate properly the advantages and disadvantages of each in a small series of cases performed by any one individual surgeon. Nor would it be of any value to select isolated cases from a group of individual operators. One of us[1] reviewed a series of cases at a private institution where there were only three operators. It is with this thought in mind that a statistical review was made of 550 consecutive hysterectomies performed by forty-eight different surgeons plus the resident staff, the latter working directly under supervision and closely following the technique of the attending gynecologist.

Before presenting the series of cases it would seem advisable briefly to review the current literature, especially those papers in which a relatively large series of cases are analyzed.

Siddall and Mack[2] have collected a series of cases from the literature comprising 7,795 subtotal and 4,559 total hysterectomies. To this series they added 1,141 cases of subtotal and 235 cases of total hysterectomy from one hospital. If to this group is added a series of cases reported by Gardiner and Kretzschmar,[3] the total number of cases reported will be 9,606 subtotal hysterectomies and 4,985 total hysterectomies.

The combined ratio of total to subtotal is approximately 1:2 and the combined mortality is 3 per cent in the subtotal group and 3.56 per cent in the total group. This we believe to be a fairly representative group of cases seen throughout the country in well organized hospitals of accredited standing.

A further analysis of the individual reports bring out some extremely interesting facts. From the analysis in Table 1 it appears obvious that the greater the number of total hysterectomies done in any one series, the lower the mortality (with the exception of the series reported by Burch and Burch). It would seem only logical to assume that the greater skill developed by performing an increasing number of total hysterectomies would diminish the mortality percentage on a

TABLE 1

	Subtotal Hysterectomies		Total Hysterectomies	
	Number	Per Cent Mortality	Number	Per Cent Mortality
Masson	217	1.8	229	1.3
Nelson	122	3.2	476	2.9
Fullerton and Faulkner	609	4.4	1,078	4.1
Burch and Burch	166	4.2	32	3.1
Mayo and Mayo	3,085	1.2	1,588	1.8
Greenhill	1,857	4.47	551	4.72
Nead and Bill	1,739	2.1	605	3.1
Siddall and Mack	1,141	2.6	235	6.4
Gardiner and Kretzschmar	670	3.1	191	4.7
Total	9,606	3	4,985	3.56

TABLE II

WOMAN'S HOSPITAL SERIES

	Subtotal	Total
Number of cases	350	200
Youngest	23 years	23 years
Oldest	68 years	62 years
Average	38.5 years	39 years
Number of operators 48		

* From Woman's Hospital, Detroit, Michigan.

technical basis. It would certainly not deter the surgeon from the procedure because of relative unfamiliarity, provided other considerations were equal.

The average age in both groups is for all practical purposes the same. The number of individual operators is sufficiently large and varied in gynecological experience and skill to permit a fairly accurate general interpretation of the subsequent statistical tables.

Table III
SYMPTOMOLOGY

	No. of Cases	Percentage
1. Bleeding	242	48.0
2. Abdominal pain	170	30.9
3. Abdominal mass	61	11.1
4. Backache	40	7.3
5. Frequency	20	3.6
6. Procidentia	6	1.1

The symptoms presented were mainly bleeding, abdominal pain and abdominal mass. These symptoms were present either singly or in combination in approximately 90 per cent of all cases. The most common symptoms were vaginal bleeding—48 per cent; abdominal pain—30.9 per cent; and abdominal mass—11.1 per cent. Other less constant symptoms were backache, frequency and procidentia.

Table IV
ANESTHESIA

	Subtotal		Total		Combined	
	No.	Per Cent	No.	Per Cent	No.	Per Cent
Gas-ether	307	87.4	150	75	457	83
Spinal	35	10	21	10	56	10.2
Avertin	6	1.8	29	14.5	35	6.3
Gas	2	0.76	0		2	0.36
Average duration of operation	1 hour 15 minutes		1 hour 18 minutes			

In the entire group the anesthetic use in 83 per cent of the cases was gas induction and ether. Spinal anesthesia was used in 10.2 per cent; avertin as a basal anesthetic in 6.3 per cent, and in only two cases, or 0.36 per cent, was gas alone used. It is interesting to note that in the choice of anesthesia, the percentage of the individual anesthetics employed in each group is practically alike, except in the use of an avertin basal anesthesia. This was used in five times as many total as subtotal cases. Whether the use of avertin had any effect on the immediate postoperative morbidity one is unable to state definitely. It should, however, be noted that the patients required closer observation during the first twelve postoperative hours.

As to the duration of operation, only three more minutes on the average were required to complete the operation of total hysterectomy as compared to subtotal. Certainly the time element is of no great significance, and should not be an influencing factor in deciding against total hysterectomy.

Table V
ADNEXAL AND ADDITIONAL OPERATIONS

	Subtotal		Total		Subtotal and Total	
	No.	Per Cent	No.	Per Cent	No.	Per Cent
1. Bilateral salpingo-oöphorectomy	100	28.9	76	38	176	32
2. Unilateral salpingo-oöphorectomy	70	17.1	66	33	136	24.7
3. Salpingectomy	14	4	4	2	18	3.3
4. Oöphorectomy	22	6.2	6	3	28	5.1
5. Appendectomy	208	59.7	108	54	316	57.4
6. Dilatation and curettage	24	6.4	10	5	34	6.2

In the entire group additional adnexal operations were done in 65.1 per cent. In the subtotal group alone, these operations were done in 56.2 per cent. Of these 35.1 per cent had all ovarian tissue removed. In the total group 76 per cent had adnexal operations, but only 41 per cent had all ovarian tissue removed. Although 19.8 per cent more of the total group had adnexal operations, only 4.9 more in this group had all ovarian tissue removed.

The percentage of appendectomies in both groups is higher in the subtotal group, but since these were prophylactic measures one can attach no significance to this.

In the subtotal group 8 per cent of the cases developed complications, while in the total group 13.5 per cent developed complications. Patients in the total group had 19.8 per cent more adnexal operations as compared to the subtotal group. This undoubtedly has some direct effect on the morbidity, although one is unable to make this assertion upon sound scientific basis.

Peculiarly enough, there were five cases of postoperative shock in the subtotal group. Obviously then, in this series postoperative shock is not an important deciding factor, since this complication occurred at least as often in both types of operation.

However, when we consider pulmonary embolus, it is found that whereas only two cases in the subtotal group were involved, in the total group five had this serious complication. Some writers have reported more cases of pulmonary embolism in the subtotal group and they believe that there is less venous trauma in the total group where more of the veins are excised.

In the subtotal group one patient developed a troublesome cystitis and in one case the bladder was opened at the time of operation. In the total group, while there were also only two complications involving the genitourinary tract, they were of a more serious nature, a bladder fistula and a bladder and rectal fistula. Although much mention is made of the fact that the total operation endangers the bladder, ureters and rectum, in this series this complication was encountered only in two cases, or 1 per cent, and not in a single instance were the ureters damaged.

Minor postoperative vaginal bleeding occurred in two of the total group, both on the second postoperative day. There was none in the subtotal group.

While only one case of pelvic peritonitis is listed in the subtotal group, it is well to note now that in the mortality table there are two cases of generalized peritonitis in each group with fatal termination

The other complications listed are not peculiar to either type of operation, but rather to abdominal surgery in general, and do not hold any special significance.

The average stay in the hospital was only 0.6 day more in the total group than in the subtotal group.

The temperature peak in both groups was reached on the first or second postoperative day and subsided gradually to normal on the fourth or fifth postoperative day. The average highest temperature was only 0.2 per cent of a degree higher in the total group than in the subtotal group.

TABLE VI

MORTALITY—LISTED CAUSES OF DEATH

	Entire Series	Total	Subtotal
Peritonitis	4	2	2
Evisceration	2	0	2
Malignancy with metastasis	1	0	1
Pulmonary embolus	1	0	1
Paralytic ileus	1	0	1
Cardiac failure	2	2	0
Anuria with nephritis	1	1	0
Total	12	5	7

In the entire group of 200 total hysterectomies there were only two deaths due to peritonitis, or 1 per cent mortality. This mortality is not particularly high in view of the pathology of the tissues involved, and does not necessarily bear out the contention that opening the vaginal vault predisposes to peritonitis. An equal number of fatal peritonitis cases was encountered in the subtotal group, although the percentage of mortality here was somewhat lower, 0.6 per cent. The other fatal complications cannot be classed as contraindicating either one or the other type of hysterectomy. Thus two fatal cases of evisceration occurred in the subtotal group, and two cardiac deaths in the total group. The death due to anuria with nephritis is interesting, more from the view of previous antiluetic therapy and its relation to anaesthetic rather than to the choice of surgical procedure. There was no damage to the ureters during the surgical procedure, and so it cannot be considered as a complication peculiar to the procedure carried out.

DISCUSSION

From the cases analyzed in the literature and the series of cases from Woman's Hospital, certain factors lend themselves to generalization.

The mortality and morbidity in total hysterectomy is slightly higher than in subtotal in a general hospital with a large group of operators. The total operation as such must therefore be considered a more formidable procedure. Yet it has been shown that the more frequently this procedure is employed and the more familiar

the surgeon becomes with the operation, the lower the mortality.

To guard against the possibility of coexisting carcinoma, a routine vaginal preparation is of the utmost importance. Under anesthesia and ideal operating room facilities it is only a matter of a few minutes to reëxamine the patient and whenever indicated to do a diagnostic curettage and frozen section.

It has been stated that the cervical stump is necessary to act as a keystone of the arch to prevent prolapse after hysterectomy. Actually this is a rare occurrence following total hysterectomy providing the round ligaments are properly suspended. With regard to leucorrhea and backache, Nelson[1] has encountered less difficulty with these troublesome sequelae in his total group than in the subtotal group, and Gardiner and Kretzschmar[3] report a 7 per cent higher incidence in the subtotal as compared to the total group.

It is not the purpose of this discussion to advocate the use of total hysterectomy rather than subtotal and vice versa. Rather it would be well to emphasize that in view of the above considerations total hysterectomy is not so formidable as to greatly limit its more general utilization. The general physical condition of the patient, the pathology present and the age must always be influencing factors, even to the surgeon who is equally familiar with both procedures.

Richardson[7] refers to the types of cases that are suitable for subtotal hysterectomies, as:

1. Those women requiring hysterectomy for benign disease who possess perfectly normal cervices.
2. Those instances in which the operative hazard compels the execution of conservative surgery.
3. Those cases where, for good and sufficient reasons, it is of paramount importance to preserve the menstrual function.
4. Most cases requiring hysterectomy during pregnancy.

These are factors well worth pondering. It would also appear advisable to consider the endocrine status of the patient following subtotal or total hysterectomy and its relation to the onset of menopausal symptoms. It has been shown that the normal woman in going through her menopause has an increase in her blood prolan and a decrease in blood estrin. Marx, Catchpole and McKennan,[4] working with a series of twenty-one clinical patients, after a careful and detailed study showed that the patient who had a total hysterectomy is more likely to undergo her menopause at an earlier date than the patient who had a subtotal hysterectomy. It is their belief that the uterine endometrium elaborates a catalytic principle acting on the pituitary-ovarian hormonal mechanism, regulating its normal balance and functional harmony. This phase of the problem is extremely interesting, and like all endocrine studies, additional work with a larger group of patients should be conducted before definite conclusions can be drawn.

CONCLUSIONS

1. A series of 550 abdominal hysterectomies from the Woman's Hospital is presented. The cases covered a period of 4½ years and included 350 subtotal and 200 total hysterectomies.
2. This series shows a slightly higher mortality and morbidity for the total group. It must be borne in mind, however, that the difference is not sufficiently great to justify a decision against this procedure where there are indications for its use.
3. The total hysterectomy should be more frequently done in those cases where the cervix shows signs suspicious of premalignant states. Only too frequently is a malignancy of the cervix encountered shortly after subtotal hysterectomy. Undoubtedly there was a coexisting early malignancy of the cervix at the time of operation.
4. Clinical follow-up in the total group with relation to the endocrinological disturbances caused by removal of the cervical stump needs further study.
5. This problem should not be considered in the light of total versus subtotal hysterectomy.

It would be much better for the surgeon equally familiar with both procedures to employ the one which appears to be indicated in each individual case.

REFERENCES

1. Nelson, H. M. *J. Michigan M. Soc.*, 28: 860, 1929.
2. Siddall, R. S., and Mack, H. C. *Surg., Gynec. & Obst.*, 60: 102, 1935.

3. Gardiner, S., and Kretzschmar, N. R. *J. Michigan M. Soc.*, 36: 13, 1937.
4. Marx, R., Catchpole, H. R., and McKennon, B. J. *Surg., Gynec. & Obst.*, 63: 170, 1936.
5. Samuel, H., and Edlavitch, E. S., *Obst. & Gynec.*, March, 1933.
6. Goodall, J. R. *Am. J. Obst. & Gynec.*, 26: 556, 1933.
7. Richardson, E. H. *Am. J. Obst. & Gynec.*, 32: 641, 1936.
8. Polak, J. O. *J. A. M. A.*, 75: 578, 1920.
9. Baldwin, J. F. *Am. J. Obst. & Gynec.*, 75: 262, 1917.
10. Read, C. P., and Bell, A. C. *J. Obst. & Gynec. Brit. Emp.*, 40: 749, 1933.
11. Greenhill, J. P. *Am. J. Obst. & Gynec.*, 24: 183, 1932.
12. Fullerton, N. D., and Faulkner, R. I. *J. A. M. A.*, 95: 1563, 1930.
13. Moench, G. L., and Holt, H. *Am. J. Obst. & Gynec.*, 22: 199, 1931.

Prolan (from the chorionic cells of the human pregnant uterus), the gonadotropic hormone characteristic of pregnant-mare serum (likewise probably secreted by chorionic or endometrial cells), and the gonadotropic hormones of neoplasms such as hydatidiform mole, chorionepithelioma, and testicular tumor (from the cells of the neoplasm) . . . appear not to be secreted by the hypophysis.

From—"The Physiology and Pharmacology of the Pituitary Body," vol. II, by H. B. Van Dyke (University of Chicago Press).

TREATMENT OF DYSMENORRHEA*

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A WOMAN is said to be suffering from dysmenorrhea when the subjective manifestations of the menstrual period are so exaggerated that she complains of marked pain or discomfort in the lower abdomen and pelvis, thighs, or sacral region. Severe dysmenorrhea is often accompanied by such symptoms as headache, eye-strain, contraction of the field of vision, cutaneous eruptions (acne and eczema), and neuralgic conditions (toothache and joint pains).

We will consider here only the treatment of severe dysmenorrhea. The ordinary cases where almost any antispasmodic or analgesic affords relief are not within the scope of this paper. Our results were obtained by the use of corpus luteum extract in oil, and anterior pituitary-like hormone made from pregnancy urine. The treatment of severe dysmenorrhea has never been uniformly successful.

Boynton used calcium gluconate and obtained complete relief from abdominal pain, leg cramps, paresthesias and nausea in thirty-three out of forty-nine cases treated. The calcium gluconate was given in 60 gr. doses daily for two weeks before the onset of the menstrual period; when viosterol was added, 30 drops daily was given.

De Benedetto claims very good results from the use of bellergal, said to contain phenobarbital 0.02 Gm.; ergotamine tartrate 0.00003 Gm.; levorotatory belladonna alkaloids 0.0001 Gm. The patients were directed to take orally one tablet with water every two hours but not to exceed five or six tablets in any one day. Treatment began with the onset of pain and was continued, if necessary, during the first day or two or throughout the entire period. Rest and particular attention to elimination were observed. In thirty cases, the menstrual pain was completely abated within six hours after the first administration of the tablets. In twelve cases, it was necessary to continue the treatment during the first two days of the menstrual flow. Treatment was required in ten cases throughout the entire menstrual period. Headache, in varying degrees of severity, was also completely relieved by the therapy.

Cary has used a new non-narcotic synthetic antispasmodic agent which apparently has the useful qualities of both atropine and papaverine with the disadvantages of neither. This is the 3-diethyl-amino-2, 2-dimethyl-propanol ester of tropic acid and is marketed under the trade name of syntropan in the form of 50 mg. tablets. In animal experimentation, Fromherz tested the antispasmodic action of syntropan on the isolated rabbit intestine against the stimulating effect of acetylcholine, and on the isolated rabbit uterus against the action of epinephrine. He found that the relaxing effect upon the intestine is five times stronger than that of papaverine. Only very small doses were needed to produce complete relaxation of

* Read before the Brooklyn Gynecological Society, April 1, 1938.

the intestinal musculature following acetylcholine stimulation. Syntropan acted similarly to atropine as an inhibitor of the parasympathetic division of the autonomic nervous system, and thus tended to reduce excitatory states of smooth muscle. Pharmacologic studies of Fromherz and others demonstrated that in therapeutic dosage the antispasmodic effect of syntropan corresponded to that of atropine but that its use was free from disagreeable by-effects on circulation, the pupil and glandular secretion which too often attended the administration of atropine.

In fifteen cases, pain varied from moderate to severe. Single, married, and widowed women were included in the series. Ages ranged from 27 to 40 years. All cases were apparently benefited, 60 per cent returning to normalcy, 40 per cent being improved. Medication was instituted twenty-four hours preceding onset of the menstrual period and then continued for the first day of the period. Remission of localized uterine pain was not the sole benefit derived from treatment; other constitutional symptoms disappeared.

Altschul reported twelve cases of dysmenorrhea occurring mostly in patients below par physically, underweight, and often with low basal metabolism, which he treated with insulin, 7 to 10 units daily, either at the time of the pain or over a period of three days prior to the menstruation, with almost total relief from pain in ten, partial relief in one, and relief in one of two periods in the twelfth case. Blood sugars in these patients were normal.

Watson treated 105 cases of dysmenorrhea with the placental extract, emmenin. Of these, forty-nine were completely relieved, the benefit lasting as long as six months after cessation of treatment in twenty-one of these cases. In twenty-seven patients relief was experienced to a degree which enabled them to overlook the remaining discomfort. In twenty-nine cases there was no amelioration obtained. Best results were gotten when three teaspoons of emmenin were given two weeks prior to the menstrual period. Emmenin is a powerful estrogenic substance present in acetone extracts of human placentae, alcohol-soluble, ether-insoluble, active orally, though relatively inert in oöphorectomized animals.

Witherspoon obtained relief from dysmenorrhea in thirteen of seventeen patients treated by injection of 250 rat units of follutein intramuscularly daily, for three to four days previous to the expected flow. The luteinizing principle found in the urine of pregnant women is believed responsible for the results obtained.

Israel, reporting on thirty-nine cases of severe dysmenorrhea, obtained relief in four of ten cases with antuitrin-S, 200 rat units being given every other day for from two to three months; in three of ten cases with emmenin, one tablespoonful (75 day-oral units) three times a day from three to five months; in one of six cases using progynon 600 rat units three times daily for three to four months; and in ten of thirteen cases using 5,000 to 10,000 rat units every fourth day for three to four months. In the last group, four were relieved for at least two to four months after withdrawal of the medication and six while continuing the injections, but none were permanent cures. Up to 200,000 and 3,200,000 rat units of progynon were tolerated well by this last group of patients.

Work with benzedrine for the relief of dysmenorrhea has already been begun, without any published reports as yet of the results.

Bauer administered pitressin to six patients with dysmenorrhea, 0.5 c.c. at the beginning and during the period of the pain, and obtained partial or complete relief. Immediately after injection there was a temporary increase in the cramps, probably, he stated, due to the small amount of the oxytocic fraction in the preparation. The blood pressure was carefully watched during this procedure and at no time was a dangerous rise observed. He does not state the effect on the gastrointestinal system. Reynolds showed that uteri in a state of contraction induced by

calcium became relatively quiescent three or four minutes after injection of 0.5 c.c. of pitressin and were refractory to subsequent injections of calcium or pitocin unless the uterus was again motile. Moreover, it was frequently observed that pitressin for a time abolished the spontaneous contractions of the uterus and rendered this organ transiently refractory to both calcium and pitocin. Bauer used antuitrin-S in several cases, in 1 c.c. doses, at various intervals during the second half of the menstrual cycle, and obtained relief of moderate degree in one case.

Emge found theelin to be of aid in dysmenorrhea in only six of twelve cases, and of these four improved only moderately. He believes that vaginal suppositories containing acetanilid, sodium borate, zinc borate and hydrastin, in glycerogelatin, brought the best results. Thyroid gave relief in eight cases, partial in six, and no benefit in four. Thyroid was given after a basal metabolism test, and the clinical evidences of the patient, namely, fatigue, obesity, and sluggishness showed the presence of thyroid insufficiency.

Kennedy, using progynon, sistomensin, and ampacoids and ovacoids, obtained complete relief in ten cases, improvement in thirty, and no amelioration in ten. Dosages were not stated. Occasionally pessaries were substituted for the injections. He based his reasoning on the fact that ovarian hormones aid restoration of the cervical ganglia of the cervix to normal. Previous investigation showed that after ovariectomy the cervical ganglia suffer in that the Nissl granules are lysed, the cells shrink, while the number of chromaffin cells always associated with the ganglia diminish markedly. Restitution of the original picture was obtained by the injection of estrin, and maintained by repetition of the administration.

Tunis reported a series of seventy-eight cases in which he used menformon, an estrogenic preparation. These women had dysmenorrhea associated with hypoplastic uteri. Excellent relief was obtained in sixty-five cases; eight patients were improved and five were refractory to treatment, which consisted in six injections of 1,000 units each.

Hacker reports a case in which excellent results were obtained from corpus luteum, even when given by mouth. This relief continued for months at a time, but with recurrence of pain sooner or later. Following a suspension operation for retroversion of the uterus, there was relief for a year. The use of 1,000 Allen-Doisy units ten days prior to menstruation for three months brought complete relief.

The uterus of the experimental animal is in a state of motility, and definite contractions can be recorded when the animal is in a state of estrus. Under the influence of the animal's own corpus luteum the uterus becomes quiescent, no contractions can be elicited, and it is refractory to pituitrin. Knaus' and Reynolds' work concur on this point. On the day before the onset of menstruation the uterus reverts to its preovulatory behavior, which may help explain some dysmenorrheas.

Wintz used sistomensin in 125 cases of dysmenorrhea, giving 5 c.c. daily for several days prior to the menstruation. He obtained complete relief in eighty-two cases, improvement in fifteen, and no effect in twenty-eight.

Elden and Wilson, using progestin, obtained complete relief from dysmenorrhea in 47 per cent of seventeen patients, partial in 12 per cent and none in 41 per cent. The patients were normal women with normal pelvic findings. The dosage used was $\frac{3}{25}$ to $\frac{6}{25}$ units, the amount being given in divided or single doses three to six days before the menstrual flow or before the onset of pain. It was given at this time because of Knaus' observation that the uterus reacts to pituitrin the day before the onset of bleeding and because the hormone is in oil and therefore slowly absorbed. There was no delay in the onset of the menstrual cycle, and apparently no change in the duration of the flow. Several of the patients who

experienced no relief received larger doses, up to 1 unit. Two of these patients had subsequent presacral sympathectomies with complete relief. Two patients received estrogenic hormone up to 50,000 international units with little effect.

Hisaw and Campbell have used the preparation corporin, a pure chemical product of the corpus luteum hormone alone. They confined their therapy to a definite class of dysmenorrhea patients with normal genital development and placement. Patients with infantile genital development, cervical stenosis and improper drainage, ovarian abnormalities, or other extragenital conditions which might cause the periodic pain, were excluded from their series. Of five patients, four found complete relief and the fifth moderate ease. Their dosage was 5 to 8 rabbit units daily for five days prior to the menstrual period. Two patients flowed more than ordinarily after several series of injections.

We are here reporting ten cases of severe dysmenorrhea, not apparently associated with any specific cause, the so-called essential dysmenorrhea. Histories of several cases are appended.

M. L., age 24, single, had always suffered with severe pain and cramps at the menses, which began at 12 years, were moderately heavy in amount of flow, and recurred regularly each month. The patient was bedridden at least every other menstruation. Rectal examination revealed no pelvic abnormalities. Estrin excretion was 32 units per day with a strong positive pituitary prolan reaction.

This patient received ½ international unit of progesterone every other day for ten days prior to the period, and had absolutely no pain. The following month she was given the same treatment, but the period being delayed, the treatment was continued for about 3½ weeks. This period was also devoid of any pain. There was delayed flow the following month and the same excellent result from treatment. The patient has required no further injections. The menses have regulated themselves, and nine months later there had been no recurrence of the severe dysmenorrhea.

R. P., age 28, married, gave a history of severe dysmenorrhea for about ten years, both prior to and since her marriage three years ago. A previous dilatation and curettage effected only a very temporary beneficial result. Her periods were regular, of five days' duration, and accompanied by such pain that the patient took either several injections of morphine, or a large amount of alcohol to deaden the pain.

This patient was very coöperative and intelligent. Anterior pituitary-like hormone, 100 units, was given three times weekly for one month, with marked exaggeration of the symptoms. Since it was possible to gauge accurately the onset of cramps, ½ to 2 units of progesterone was given about twelve hours prior to the pain with almost entire relief. This was repeated each month for seven months; then two months were allowed to elapse without treatment, and without any pain. It was then necessary to renew treatment. An endometrial biopsy was attempted premenstrually and a tight cervical stenosis was found. A glass stem pessary was inserted, which resulted in disappearance of pain for a few months. Scant endometrium was obtained on curettage and was reported as progestational in character.

G. W. age 26, married fifteen months, gave a history of dysmenorrhea for ten years. Her periods were regular, but with menorrhagia and dysmenorrhea. The right breast was larger than the left, and a tumor had been removed from it a year before. The uterus had a small fibroid in its lower segment. The patient was sterile and biopsy of the endometrium showed no progestational changes. Hormone determinations disclosed 8 units of estrin, and a negative prolan reaction. She was given ⅕ unit of progesterone every three days for ten days premenstrually during three months. No dysmenorrhea or menorrhagia has occurred since. She was delivered of a normal child nine months after the cessation of treatment.

C. P., age 19, complained of dysmenorrhea since the onset of menses, which, however, occurred regularly. The uterus was normal upon rectal examination and no adnexal pathology was found. The dysmenorrhea increased with time. Acne, comedones, and mild hirsutism were present. Basal metabolism was minus 10. Estrin and prolan determinations were normal. Pain in the breasts was present twenty-four hours premenstrually, and abdominal pain for three days before bleeding. Progesterone

1/5 unit was given every 3 days, ten days premenstrually for four months. There has been no dysmenorrhea since. The acne has disappeared. There was no effect on the hirsutism.

A. M., 31 years old, married, had one child five years previous. This patient had menorrhagia for three and one-half years after childbirth, and in addition, dysmenorrhea for the past one and one-half years. Hormone studies were normal. Treatment with 1/5 unit of progesterone every three days for ten days premenstrually was instituted. The menorrhagia and metrorrhagia stopped in two months, the dysmenorrhea in four months. In fourteen months since cessation of treatment there has been no return of pain or excess bleeding.

F. B., age 25, single, had a history of increasing dysmenorrhea for five years. Rectal examination revealed no pelvic pathology and hormone determinations were normal. Anterior pituitary-like hormone given in 1 c.c. doses every other day during the last half of the cycle for four months, gave relief from the severe pain and it was possible to discontinue treatment for several months without any recurrence.

R. L., age 21, single, had dysmenorrhea since onset of menses at the age of 14. The genitalia were apparently normal. Anterior pituitary-like extract injected, 1 c.c. twice a week during the second half of the cycle, brought about complete relief. Treatment was continued for three months, and pain has not recurred since discontinuance of medication over a year ago.

SUMMARY

Almost all types of hormones have been used in treating dysmenorrhea, with good results in a considerable percentage of cases.

Hormone determinations have only occasionally been of value as a determining factor in treatment.

Endometrial biopsy is a valuable aid in the determination of the state of uterine and ovarian activity.

In our series of ten cases, eight were benefited either partially or completely by administration of corpus luteum hormone, and two were aided by anterior pituitary-like hormone. One patient helped by progesterone was definitely made much worse by anterior pituitary-like hormone.

We wish to express our appreciation for the coöperation accorded us, and the material supplied for the above work, by the Schering Corporation (Proluton—corpus luteum hormone), Winthrop Chemical Co. (Antophysin—anterior pituitary-like hormone), and E. R. Squibb (Follutein—anterior pituitary-like hormone).

REFERENCES

Boynton, R. E. *Am. J. Obst. & Gynec.*, Feb. 1934.
De Benedetto, G. *M. Rec.*, Sept. 4, 1935.
Cary, F. S. *Urol. & Cutan. Rev.*, Aug. 1936.
Altschul, A. *J. A. M. A.*, April 18, 1936.
Watson, M. C. *Canad. M. A. J.*, June, 1935.
Witherspoon, J. T. *Endocrinology*, July-Aug., 1935.
Israel, S. L. *J. A. M. A.*, May 16, 1936.
Bauer, L. E. *J. Michigan M. Soc.*, Aug. 1934.
Emge, L. A. *Calif. & West. Med.*, Dec. 1933.
Kennedy, W. P. *Brit. M. J.*, April 23, 1932.
Wintz, H. *Münchcn. med. Wchnschr.*, April 3, 1931.
Elden, C. A., and Wilson, K. M. *Am. J. Obst. & Gynec.*, July, 1936.
Campbell, R. E., and Hisaw, F. L. *Am. J. Obst. & Gynec.*, March, 1936.

THE EFFECT OF A BUFFER OF MANDELIC ACID AND ESTROGENIC HORMONE IN THE TREATMENT OF TRICHOMONAS VAGINALIS VAGINITIS*

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IT would seem superfluous to add another contribution to the long list of articles concerning the eccentricities and behavior of the trichomonas vaginalis, unless very definite data of etiologic and therapeutic import could be presented. The sole purpose of this paper is to make a few observations regarding the rationale of therapeutic application in this infection and add another chemical agent to the already long list.

The majority of investigators in this field seem to believe that the source of the infection is from the intestinal tract. The protozoan is harbored there, apparently in an innocuous state, as no pathologic condition of the intestines has been attributed to it. The close proximity of the rectum to the vagina makes for easy transmission of the organism. When entrance is obtained the behavior of the trichomonad may be harmless, but it may induce, under proper conditions, an inflammation of the vagina. The inflammatory reaction produces small superficial ulcerative lesions of the vaginal walls and cervix (portio) that result in a profuse yellowish discharge, which microscopically consists of numerous pus cells, a limited number of epithelial cells, and many trichomonads in an active state of motility. The discharge as a rule is quite profuse, may have a disagreeable odor, and produces an irritation and itching of the vulva that continues in spite of the ordinary vaginal douches taken. To the patient suffering from this infection is imparted not only physical discomfort, but a mental depression due to the sensation of uncleanliness.

Treatment of this condition has endeavored to destroy the organism through the use of different chemical agents. Many preparations have been used, and while they have been helpful and have effected cures, there is much left to be desired as to a definite specific outline in establishing better results.

The trichomonad is sensitive to most chemical substances and seems easily destroyed following the usual therapeutic measures. However, in a large percentage of cases, recurrences are encountered. This is quite characteristic and occurs in spite of thorough investigation of the sources of reinfection, from the urethra and bladder urine and the semen of the consort. Much has been written concerning the treatment of recurrent attacks, which really seems to be the crux of the problem.

Some underlying factor of a biochemical nature is evidently at fault. Along these lines, many observations concerning the behavior of the trichomonad in the vagina have been made. In the first place, the vagina can harbor these organisms without pathologic reaction of the tissues. The vagina also may apparently be free of the trichomonads, but as menstruation approaches positive specimens are seen. In view of this observation it has been thought that the trichomonads were harbored in the uterine cavity and were active just before and during the menstrual flow. I have taken many specimens from the uterus and cervix of such patients and have failed to find a positive specimen.

We do know that the reaction of the normal vagina is acid, with a *pH* of 4 to 4.5;

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that microscopically a smear will show over 95 per cent epithelial cells, some mucus, occasional pus cells, and many Döderlein bacilli.

Interesting observations have been made by Dobszay[1] as well as by Cruickshank and Sharman[2] concerning the vaginal secretions of infants and children. In the newborn the reaction of the vaginal secretions was found to be acid and the Döderlein bacillus was evident. This state continued for seven to ten days. Their explanation for this reaction was assumed to be due to the continued action of the maternal estrogenic hormone absorbed through the placental circulation in utero. After the tenth day following delivery, the estrogenic hormone having been excreted, the vaginal secretions became alkaline without the presence of the Döderlein bacillus and continued in this state through childhood. At puberty and adult life the acidity and the presence of the Döderlein bacillus were the prevailing characteristics of the vaginal secretions. At and following the menopause there is a reversion to the prepubertal condition of alkalinity. The presence of the Döderlein bacillus is important, as its function seems to be to maintain the normal acid reaction of the vaginal secretion by its action upon the glycogen of the vaginal epithelium. We also know that in trichomonas vaginitis there is a complete disappearance of the bacillus in pronounced and severe cases. Only a few remain in the the milder types, or in patients rapidly improving under treatment. In the severe forms of the infection the reaction of the vaginal secretion leans toward the alkaline side with a *p*H of 5 to 6.5 and sometimes 7.0.

According to Karaky,[3] this is due to a deficiency of glycogen in the vaginal epithelium. Normally the Döderlein bacillus acts upon the glycogen to produce lactic acid, thus maintaining the normal acidity of the vagina. I am much in accord with Karaky. In the successful treatment of these infections, the basic aim of therapy should be to lower the vaginal *p*H, thereby increasing and establishing a higher acidity of the vaginal secretions. To produce this favorable condition, the introduction of a substance having a *p*H of 2.0 to 3.0 would overcome the alkaline tendency and bring about an unfavorable soil for the growth of the trichomonad and associated bacteria.

In selecting such a chemical it seemed necessary to choose one that was not irritating to the vagina. As mandelic acid seemed to be so successful in the treatment of renal and bladder infections by rendering the urine highly acid, destroying the bacterial flora and preventing their propagation and growth, we decided to apply it to this type of vaginal infection. It has been a much discussed point whether the Streptococcus subacidus is the real culprit in producing the local lesions and symptoms in trichomonad infestations, as it is so frequently encountered in specimens and cultures from the vagina of those suffering from the disease. This was another reason for trying mandelic acid in the condition. This drug has been markedly successful in streptococcus infections of the urinary tract. It seemed desirable to endeavor to maintain the lowered *p*H of the vaginal secretion following the therapy with mandelic acid buffer.

The administration of estrin to patients with senile vaginitis is practically a specific for the healing of the superficial ulcerations. It was thought that it might aid in a similar manner in trichomonas vaginitis lesions, at the same time increasing the acidity of vaginal secretions and antagonizing the growth of the organism. In conjunction with sodium mandelate insufflations, therefore, injections of 30,000 international units of estrogenic substance were given three times weekly. This idea was suggested by the work of Lewis and Weinstein[4] in their investigations of gonorrheal vulvovaginitis of children. They found that the normal vaginal reaction in children was alkaline (*p*H 7.2) and that the secretions were free of the Döderlein bacillus. In gonorrheal vaginitis the reaction showed a *p*H that was neutral. The estrogenic

hormone caused the vaginal reaction to become acid, with disappearance of the gonococci in a surprisingly short time.

To evaluate and apply these findings in patients with trichomonas vaginalis infestation, a group of patients were selected who suffered from severe and pronounced symptoms of vaginitis, profuse yellow discharge and aggravated pruritus. Microscopic examination showed numerous motile trichomonads. The vagina and portio gave evidence of the characteristic lesions of superficial ulcerations that bled easily when wiped with a cotton ball. Biopsies from these areas were reported as typical. The reaction of the vaginal secretions was neutral or slightly acid, ranging from 5.5 to 7.0.

These patients received 30,000 I. U. of estrogenic substance three times weekly, given intramuscularly in the gluteal region. No other form of therapy was prescribed except a daily cleansing vaginal douche of normal saline solution. The vaginal reactions were carefully taken and the secretions examined at each treatment. These patients were observed over a period of months and in every instance there was a cessation of the vaginal discharge and complete disappearance of the pruritus with resolution and involution of the vaginal and cervical lesions. This occurred when the reaction of the vaginal secretions became increasingly acid, 3.5 to 4.5 *p*H. However, in only a few instances when the vaginal *p*H became very low, 3.0 to 3.5, did the trichomonads disappear. This would lead one to infer, as other observers have, that the trichomonads of the vagina do not become pathogenic except in a soil that becomes fertile in a mildly acid or alkaline medium. I think this explains the recurrences in some instances when the vaginitis lightens up at the menstrual epoch, at which time the secretions become definitely alkaline. It may also explain how difficult it is, irrespective of the form of local therapy, to clear up these infections in the presence of a chronic endocervicitis, where the cervical discharge is alkaline. When appropriate destructive treatment is applied for the cervical inflammation, good results in the treatment of the vaginal infection can be expected.

A larger group of patients (fifteen) were selected at random from the clinic in order to study the local effect of a highly acid substance insufflated into the vagina. A buffer of mandelic acid was prepared by mixing a concentrated solution of sodium hydroxide with mandelic acid in molecular proportions. This was stirred until dissolved, then the water was evaporated. The residue was washed with ether to eliminate any remaining mandelic acid. The result was a mass which can be pulverized. To this powder of sodium mandelate was added mandelic acid and glucose in the following proportions:

4.29 Gm. of sodium mandelate
3.16 Gm. of mandelic acid
1.00 Gm. of glucose

This is well mixed and put up in receptacles of 2 Gm. each. The *p*H of this buffer is 3.0.

The vaginal secretions of the second group all showed a positive trichomonas infection of varying degrees and the reactions varied from 4.5 to 6.5. The mandelic acid powder was insufflated three times weekly, and no other form of local therapy was prescribed. These patients were carefully observed for the presence or absence of the trichomonads, the *p*H was meticulously recorded at each clinic visit, and the vaginal secretion examined. Following six insufflations, the entire group became free of the trichomonads, with a lowering of the *p*H to 3.5 to 4.5.

When this occurred the local treatment was discontinued and the patients were given injections of estrogenic hormone 30,000 I. U., three times weekly for a period of three weeks, or nine injections in all. They were observed for three weeks after the last injection.

In two instances during this period of observation two recurrences of positive trichomonas were seen, but without the subjective symptoms that were a former complaint. Careful investigation of the

urinary tract did not disclose any trichomonads in the bladder or urethra. It might also be said that in this series, examination of the rectum and specimens obtained from it did not show the presence of the trichomonas. It would seem that this protozoon acquires ready access to the vagina and lives there for a long time under favorable and unfavorable conditions, and in some instances is most difficult to dislodge.

SUMMARY

Trichomonas vaginitis is an infection induced by the trichomonas vaginalis, probably by contamination from the intestinal tract. The presence of the trichomonad in the vaginal secretion does not mean necessarily a pathogenic process because in many instances there is no discharge nor are there typical lesions of the vagina and cervix showing the presence of such an infection. When the vaginal secretion changes from the acid to the alkaline side conditions are rendered more suitable for the growth and pathogenicity of the trichomonad. This may occur when the entire body tissues are at a low resistance, as in anemia, subglandular dyscrasias, and at menstruation.

The treatment should be to build up the general body resistance by administration of iron tonics and vitamin therapy and at the same time to attempt to produce an acid reaction of the vaginal secretion, as the trichomonad does not thrive in the presence of a *pH* of 3.0 to 4.0. This can be done by insufflating the vagina with a mandelic acid buffer. The injection of estrin in 30,000 I. U. doses three times weekly is a splendid supplement in maintaining the acid reaction of the vagina, in aiding the reappearance of the Döderlein bacillus, increasing the glycogen content of the vaginal epithelium, and in destroying the trichomonads or rendering them non-pathogenic.

I should like to thank Dr. S. Piracci of the Italian Drugs Importing Company for the supply of estrogenic substance (ectofollicolina).

REFERENCES

1. Dobszay, L. V. *Deutsche med. Wchnschr.*, 61: 111, 1935; 62: 342, 1936.
2. Cruickshrank, R., and Sharman, Albert. *J. Obst. & Gynec. Brit. Emp.*, 41: 190, 1934.
3. Karaky, K. J. *Texas State J. Med.*, 32: 803–808 (April) 1937.
4. Lewis, R. M., and Weinstein, Louis. *Surg. Gynec. & Obst.*, 63: 604–643 (Nov.) 1936.

ABORTION AS A COMPLICATION OF OPERATION IN THE PREGNANT WOMAN

A PLEA FOR THE PROPHYLACTIC USE OF PROGESTERONE

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THE danger of abortion resulting as a complication of surgical procedures in pregnant women is ever present, particularly when the necessary surgery is on the internal genitalia, or on organs in close approximation to them. Various authors have cited the incidence of abortion in these cases as 4.5 per cent (Mussey and Crane), 15.8 per cent (Shallenberger), and 9.6 per cent (Casas). Several explanations have been offered for this high percentage of abortion, among which are: direct irritation of the uterus by operative manipulation (Varo, Greenhill, Shallenberger, Casas); excision of the true corpus luteum involved in an ovarian tumor or cyst; and performance of an operation at a date when the menstrual period would have appeared had the patient not been pregnant (Varo, Greenhill). Many authors have recommended, as a prophylactic measure, the use of morphine or one of its derivatives such as pantopon and dilaudid. More recently, Wilson, in writing on ovarian tumors in pregnancy, has suggested the use of progesterone as a therapeutic postoperative measure in those cases in which oöphorectomy includes the corpus luteum. This suggestion has also been voiced by Holmes.

Our study is a statistical survey of all operations excluding operative delivery, done on gravid women at the Michael Reese Hospital during a ten year period from January 1, 1928 to January 1, 1938. One hundred eight cases are reported, of which five aborted—an incidence of 4.66 per cent. This figure closely approximates that reported by Mussey and Crane, and is significantly less than the figures of Shallenberger and Cases. Of the 108 operations, ninety were intra-abdominal, and eighteen extra-abdominal. The intra-abdominal procedures were further divided into forty-eight genital, and forty-two extragenital operations.

Casas classifies genital operations as those on the genitalia whether external or internal; paragenital operations as those on organs in contiguity to the internal genitals, such as the appendix; and extragenital operations as those on structures remote from the genitalia, such as the gall-bladder, kidney, stomach, and breast. We have preferred the simpler classification of extra-abdominal and intra-abdominal procedures, as we feel that such a division bears a closer relationship to the incidence of abortion, and is not apt to be confusing.

Table 1 records our gross figures, and shows the number and percentage of the procedures most frequently done, with the number of abortions consequent to them.

Of the thirty-two appendectomies, fourteen were done for chronic appendicitis and eighteen for acute appendicitis, this latter group being complicated by one perforation with local peritonitis. In this series, one abortion occurred on the thirteenth postoperative day, although cramps had begun as early as the fifth day.

Seventeen myomectomies were performed; the indication in six was red degeneration with marked symptoms of pain, tenderness, nausea, and vomiting, and in the remaining eleven, it was twisted pedunculated subserous fibroids. There was one abortion in this group, following removal of two degenerated interstitial fibroids situated in the lower uterine seg-

Table I

	Number	Percentage	Abortions
Total cases	108		
Intra-abdominal	90		
Appendectomies	32	29.0	1
Myomectomies	17	15.7	1
Oöphorectomies and ovarian resections	27	25.0	2
Miscellaneous	7	6.5	1*
Extra-abdominal	18		
Thyroidectomies	7	6.4	0
Breast tumors	4	3.7	0
Cervical polyps	5	4.6	0
Bartholin adenectomies	5	4.6	0
Mastoidectomies	2	1.8	0
Trachelorrhaphies	1	.8	0

* Cholecystectomy.

ment. Cramps and bleeding occurred on the fifth postoperative day.

In nine of the twenty-seven ovarian operations, no mention of the corpus luteum was made, but in five of the remaining eighteen cases, this structure was removed. In two of these five there were abortions, one on the second, and one on the eleventh day following the operation. Table II summarizes the significant clinical data.

Table II

Abortion	Age	Parity	Stage of Pregnancy, Weeks	Operation	Indication	Onset Postoperatively, Day
1	34	2	8	Appendectomy	Mechanical appendix.	13th
2	31	2	8	Ovarian resection	Cyst containing corpus luteum.	11th
3	26	2	8	Cholecystectomy	Cholecystitis.	7th
4	29	2	8	Myomectomy and resection of corpus luteum	Bleeding corpus luteum. Twisted fibroid.	2nd
5	29	2	12	Myomectomy	Degenerated fibroids.	5th

It is of interest to note that most (sixty-nine) of the operations were done in the first trimester of pregnancy, and that the above five abortions occurred at this time. In addition, four of the five abortions resulted from surgery done eight weeks following the last menstrual period. The surgery in the fifth case of abortion was done twelve weeks following the last menstrual period. In other words, the operations were done close to the date upon which the patient would have menstruated had she not been pregnant. This is in keeping with the observations made by Varo and by Greenhill, although the exact relation of cause and effect has not been investigated.

In recent years, it has been shown by Falls, Lackner, and Krohn, that the corpus luteum hormone, progesterone, acts directly on the human puerperal uterus, inhibiting its motility. They have demonstrated its value in cases of habitual abortion. While it was formerly thought that removal of the corpus luteum very early in pregnancy would induce an interruption of the gestation due to cessation of hormonal activity, this has not proved to be the absolute rule, and it is now considered that progesterone may also be elaborated by the placenta, from the earliest stages of pregnancy (Ehrhardt, McGinty). Nevertheless, progesterone should have a place in the postoperative therapy of the pregnant woman. In only nine of the cases in this series, was the corpus luteum hormone used in the postoperative treatment. In none of these cases did an abortion occur, although the number of cases so treated is obviously too small to make any deductions. The remaining cases in which the five abortions did occur, were treated with morphine, pantopon and dilaudid, and the various barbiturates.

It has been shown by means of the intrauterine bag, that morphine increases, rather than decreases, uterine tonicity and motility. The same would probably be true of its derivatives, so that their efficacy in guarding against threatened abortion is questionable. While they may diminish body activity and pain sensation, they may also succeed in irritating an already excited

uterus. Certainly the barbiturates can have no specific effect on the pelvic viscera, being only soporific and hypnotic drugs. In progesterone, however, we have a medication which has been shown to act specifically on the uterus.

While it may be argued that the incidence of abortion in this or any other series is no greater than the normal expectancy of abortion in pregnancy (Mussey and Crane), or may be a result of the disease rather than the operation (Shallenberger), yet the fact does remain that improved surgical technique and anesthesia have diminished the frequency of abortion, and we can seek to lower it still further by the use of some preparation which inhibits uterine motility, and perhaps aids nidation. It is suggested that progesterone be used prophylactically in the postoperative therapy of gravid women in an attempt to decrease the consequent number of abortions.

CONCLUSIONS

1. Operative procedures on pregnant women, particularly in the early months of gestation, play a definite rôle in the incidence of abortion.

2. This incidence increases, as the manipulation involves the internal genitalia, especially the ovary bearing the corpus luteum.

3. It has been shown both experimentally and clinically that progesterone directly inhibits the motility of the human uterus.

4. No abortions occurred in a small series of cases in which progesterone was used postoperatively.

5. It is suggested that progesterone be used prophylactically in the postoperative therapy of pregnant women in an attempt to decrease the consequent number of abortions.

We thank the Schering Corporation for the proluton furnished for this study.

REFERENCES

1. Casas, R. J. *Rev. med. del Rosario*, 25: 426, 1935.
2. Varo, L. E. *Zentralbl. f. Gynäk.*, 50: 1964, 1926.
3. Shallenberger, W. F. *Am. J. Surg.*, 28: 582, 1932.
4. Mussey, R. D., and Crane, J. F. *Arch. Surg.*, 15: 729, 1927.
5. Frazier, C. H., and Ulrich, H. F. *Am. J. Obst. & Gynec.*, 24: 870, 1932.
6. Greenhill, J. P. *S. Clin. North America*, 7: 669, 1927.
7. Wilson, K. M. *Am. J. Obst. & Gynec.*, 34: 977, 1937.
8. McGinty, McCullough and Walter. *Proc. Soc. Exper. Biol. & Med.*, 34: 176, 1936.
9. Falls, F. H., Lackner, J. E., and Krohn, L. *J. A. M. A.*, 106: 271, 1936.

OSTEOPLASTIC REPAIR OF CRANIAL DEFECTS

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MISPLACED decompressive operations, when the ventil is not placed beneath the temporal muscle, and indiscriminate removal of fragments of bone in the treatment of depressed fractures of the skull lead to troublesome cranial defects. Surgical treatment of an extensive cranial osteomyelitis, especially in the region of the frontal bones, as well as radical surgical procedures undertaken for the eradication of infection of the frontal sinuses also contribute to the frequency of deforming cranial defects. Clinical observations have shown that the pericranium may regenerate bone to fill in a defect in the child and the adolescent, while in the adult only rounding of the edges of the defect occurs through absorption.

The indications for repair of cranial defects vary with the site of the defect. Epilepsy, motor and sensory, including visual epileptic equivalents and psychic variants are seen when the defects are situated over the parietotemporal and occipital regions. Intense headache and dizziness, especially on exertion or with a low barometric pressure, are nearly constant symptoms when the defects are located over the vertex. Cosmetic reasons for repair are given in defects over the forehead; to the uninitiated a pulsating defect over the forehead or a sunken hollow that bulges with brain with a lowering of the head is a most disturbing sight.

Since the time when J. v. Mackren implanted a piece of bone from a dog into a cranial defect of a Russian boy (the graft was subsequently removed for religious reasons) numerous methods were proposed advocating various filling materials, inorganic and organic. The inorganic implants —aluminum and silver plates, celluloid and vulcanite—are used more because of tradition than for valid reasons. They act as foreign bodies and are a constant source of irritation. They become encapsulated by inflammatory tissue and ultimately are displaced or moved about. They cause an inflammation of the underlying dura and lead to a pooling of cerebrospinal fluid between the thick dura and the brain. From time to time the skin covering the plate becomes congested, not unlike the skin overlying an encapsulated bullet.

Bone is the most frequently used organic substance. Heterogenous and homogenous bone transplants occupy a place midway between an inorganic object and an autogenous bone graft. They also behave as foreign bodies and present the additional disadvantage of a great tendency to absorption and disintegration. The contention that foreign bone transplants raise the calcium level in the blood and lead to better bone regeneration is pure speculation, to say the least.

Similar objections apply to transplants of autogenous bone boiled before use, as suggested by Naffziger.[1] When the bone is killed through boiling it cannot be considered a live autogenous graft; the roentgenograms of the implanted boiled bone flaps taken several months after the implantation, show extensive absorption of bone (Cases I and II of Naffziger). Besides, this boiling of the flap has a limited field of application, since it may be considered only in cases where a primary repair is the aim, and when the bone flap is affected by the extension of a neoplasm.

In evaluating various sources of autogenous grafts for the repair of cranial defects one must think, first, of the technical availability and adaptability of the graft and, second, of its ultimate fate years after the transplantation. The use of autogenous cranial transplants, consisting of the external table of the skull taken from an area

adjoining the defect, obviously is very limited. These grafts may be considered only in very small defects. The removal of a larger disc of bone from the external table of the skull involves technical difficulties known to everyone who tried such a removal; even with the use of a thin bladed specially curved osteotome the flap will break repeatedly and lose its value as a rigid support.

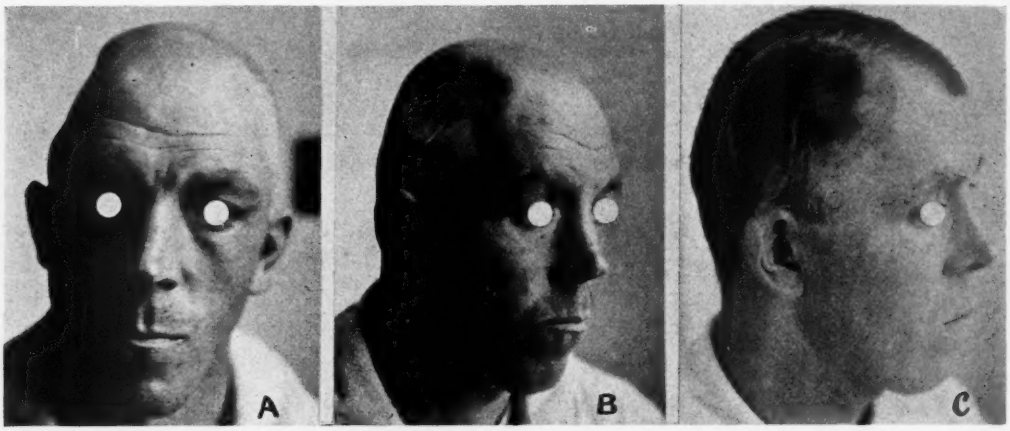

FIG. 1. A and B, showing large cranial defect resulting from poorly placed decompression done elsewhere. C, same patient, two weeks after osteoplastic repair.

A follow up of the grafts taken from the ilium, the scapula and the tibia—the most favored sites—has shown discouraging results. As a rule the transplants show marked absorption after which the defect largely reappears. Such results are to be expected when one recalls that even a primarily replaced cranial flap shows rarefaction years after the craniotomy. This is especially marked if the bone flap was stripped from the pericranium and its blood supply, as is often done in craniotomies to prevent a postoperative extradural hemorrhage. That this bone flap does not join the skull with regenerated bone but merely with fibrous tissue is well known.

This importance of the blood supply in the regeneration of the cranial bones is also seen in the ossification of linear fractures of the skull. While in children ossification of linear fractures takes place after months, in adults they become invisible only after many years. However, the portion of the linear fracture that extends through the base of the skull ossifies after a relatively brief period of time. This is explained by the difference in the blood supply of the bones of the base of the skull from that of other cranial bones. In their embryologic development the bones of the base of the skull undergo the process of chondrification and are therefore much better supplied with blood, while the bones of the vault and sides of the skull are of a membranous formation. Our knowledge of the vascular system of the bones of the skull is limited to the venous circulation. When we know better the arterial blood supply of the skull we will understand why occasionally a cranial defect is filled with regenerated bone, while in most cases bone regeneration is absent.

While the overwhelming importance of the blood supply in the regeneration of bone in fractures of long bones is well established,[2] its importance in the regeneration of cranial bones is not sufficiently appreciated. It is futile to expect incorporation of a cranial implant as in the case of bone grafts of long bones with an abundant arterial blood supply and with the physiologic effect of the muscle tone on the underlying bone and its graft. In the skull the fate of the graft depends entirely upon the speed with which the implanted bone becomes vascularized in its new surroundings. The spongy transplant covered with

its original periosteum should therefore prove the most reliable graft.

The outer half of a split rib ideally approaches this requirement. It is, besides, always available and easily adaptable. When taken from the rib in the middle axillary line the graft approaches the general convex contour of the skull. This makes bowing and bending of the graft unnecessary; because of the resiliency of the rib, bending gives only temporary results and the graft flattens out in the course of time by resuming its former shape. Remaining in intimate contact with the dura the graft causes no irritation and little thickening of it and hence fewer adhesions between the dura and the underlying brain.

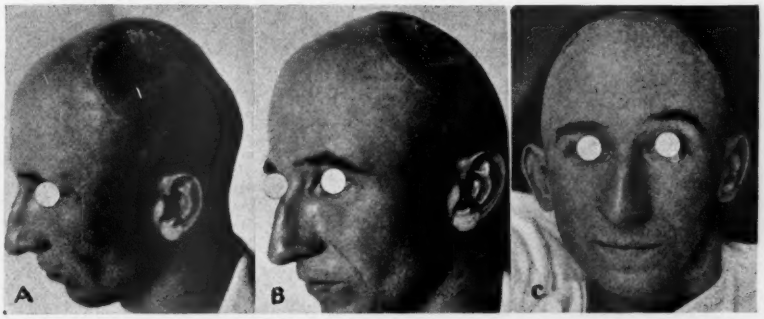

Fig. 2. A and B, cranial defect following surgical treatment elsewhere for depressed fracture of skull. C, same patient a few days after repair, showing perfect restitution of shape of skull by split ribs.

While simple in principle, osteoplastic repair by means of the split rib graft demands meticulous care of detail in the securing of the graft, its transplantation and fixation in the new location. The sixth, seventh and eighth ribs on the right side are preferred. They are approached through a curved incision which is parallel to the costal arch and is bisected in its middle by the axillary line. The exposure of the rib differs from that used in thoracotomy and thoracoplasty; one must not injure the periosteum covering the external aspect of the rib. After the external intercostal muscles are cut away, one cuts through the periosteum along the upper and lower edges of the rib, marking the line of the split. The periosteum covering the mesial aspect of the rib is pushed away somewhat to insure against injury to the intercostal blood vessels and nerves and to the pleura.

Splitting the rib is a difficult step in the operation. In the past I used a thin narrow osteotome that was introduced into the end of the exposed rib with the bevel facing mesially; the rib was thus split by the advancing chisel. Occasionally the outer half of the rib broke and, although still attached to the main graft by the periosteum, it lost its value as a supporting implant. On one occasion the chisel slid out of the split and caused a tear of the pleura. Once the osteotome slid through the internal half of the rib cutting it crosswise. I found that one avoids these complications by using a curved, small, thin-bladed bone cutting forceps instead of a chisel.

In estimating the length of the sections of the ribs to be removed one usually errs on the short side. They should measure at least 1½ inches more than the longest diameter of the cranial defect. When more than one rib is required, as in large defects, one approaches the adjacent ribs through the same curved incision. The grafts are left in situ until their new bed is prepared.

The scalp overlying the cranial defect is turned up with due regard for the arterial blood supply, for the ultimate cosmetic result and for the subsequent draining off of the blood that gathers about the graft, to prevent a hematoma that will jeopardize the ultimate result. In a large defect two or more strips of rib are used for filling in the

defect by means of a suspension bridge framework.

Before excising the extensive scar tissue underlying pericranium are sharply undermined and raised off the bone for a distance of 1 inch in length and ¾ inch in width.

FIG. 3. Extensive frontal bone defect which resulted from surgical treatment elsewhere of an osteomyelitis after radical surgery of a frontal sinus infection.

FIG. 4. Same case as in Figure 3, three years after repair with split rib grafts. Shows bony union between the grafts and skull and satisfactory density of grafts.

for preparation of the bed for the grafts, one determines the future points of support of the grafts and marks them on the shelf of the cranium. At these points the dense scar covering the bony edge as well as the The exposed bony edge is now beveled to form a bed for the ends of the ribs. It is rawed and roughened in such a manner as to secure the most intimate and close contact between the exposed diploe of the

cranium and the raw surface of the rib.

In very large defects the ends of the long grafts may be wedged and inserted into wedge-like buttresses at the ends of the gouged out bed in the skull. This will insure proper curving and bowing of the graft. In most cases, however, this tedious process may be omitted, since after the raised up periosteal flaps are turned down, tucked over the ends of the graft and sutured there the grafts are secure in their new situation.

No attempt is made to suture the dura to the graft. Great care is exercised in controlling all bleeding from the dura, the scar tissue about the cranial shelf and the turned up flap of the scalp. One avoids heavy catgut and any unabsorbable material. The scalp is then accurately sutured with a provision for drainage for twenty-four hours, to prevent an accumulation of serum that may interfere with an early adherence of the scalp and the dura to the surfaces of the graft. Snug bandaging and dependent posture for the first ten days will keep the dura in contact with the graft. The vascular scalp soon adheres to the costal periosteum, while the dura becomes attached to the spongy vascular raw surface of the graft. This promotes an early establishment of a blood supply in the grafts, with an early bony union between the ends of the grafts and the rawed bony shelf of the skull.

During the past ten years I have used this method of repair of a cranial defect in eighteen cases, in which the defects ranged between 2 and 5 inches in diameter. In five cases more than one operation was necessary before entirely satisfactory results were obtained. The final results in all cases were good; the defects were well filled and remained so years later.

At no time did any of the patients suffer later from the thoracic injury caused by the removal of the grafts. The ribs fully regenerated from the remaining half. Once a patient developed a pneumonia postoperatively on the side from which the grafts were taken. This may have been merely coincidental.

REFERENCES

1. Naffziger, H. C. Restoration of defects in skull, with special reference to management of intrinsic tumors of skull and certain types of localized osteomyelitis. *Ann. Surg.*, 104: 321–331 (Sept.) 1936.
2. Kolodny, A. Periosteal blood supply and healing of fractures. *J. Bone & Joint Surg.*, 5: 698–711 (Oct.) 1923.

DEAD TEETH AND THE ANTRUM OF HIGHMORE

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THE Antrum of Highmore has long been a controversial field. One might aptly describe it as a sort of international settlement, in which the rhinologist, the surgeon dentist and the general surgeon feel that each has some particular qualification entitling him to its operative treatment.

My comments will deal with the first two. If antral suppuration is secondary to involvement of the upper air passages and sinuses, the rhinologist is indubitably the one best qualified to render an opinion as to what procedure should be followed.

Likewise, if antral involvement is dependent upon extension from an infected or suppurating tooth root the dental surgeon, in the careful surgical removal of the offending tooth, working in a dry field with novocaine anesthesia and adequate exposure, is in an excellent position to determine what additional operative treatment is indicated. It would appear that in some selected cases, a careful study of well taken roentgenograms and a consultation and collaboration of specialists might be of advantage, particularly to the patient requiring surgical treatment of his infection.

This might be emphasized, as I have operated upon several antral floors after nasal surgery had failed to cure chronic antral suppuration. The true etiology of the antral pathology, the dead teeth, had been ignored.

A study conducted with Dr. Frank Blaisdell at Stanford University has demonstrated conclusively the extension of inflammatory and septic reactions to parts distant from dead teeth.

I have evolved an operation for the surgical removal of dead teeth which has shown a high percentage of upper tooth roots involving the antral floor, giving evidence of the real etiology of many chronic antral suppurations. Inflammatory proliferations are often direct extensions from dental pathology. Definite surgical dissection, working with direct vision and with hemorrhage under control, working only when the operative field is not obscured by blood, is essential. Injury and postoperative bacteremias from septic retention are avoided. Novocaine anesthesia and control of hemorrhage have been valuable adjuncts in obtaining this result.

Incomplete traumatizing surgery and extensive work in the presence of an acute infection are dangerous and may be the immediate cause of death. Osteomyelitis and death have followed antral surgery, particularly when an acutely inflamed sinus is curetted; bacteremia and death may follow the "pulling" of abscessed teeth, complicated by a diffuse infection. If the patient survives, postoperative fever and protracted convalescence are frequently observed. When the surrounding tissues are involved in an acute infection, incision, drainage and moist heat should be employed until the process becomes localized so that surgery may be undertaken with minimum risk to the patient.

Infective granulations must be completely removed, as retention in a sinus or a tooth socket means trouble. I find a rotated sponge is usually sufficiently effective in removing granulations from the sinus. If this method does not succeed, they must be dissected out carefully. Blindly curetting the antral cavity has resulted in injury to the lining membrane and the division or cutting of the alveolar nerves which lie between the antral membrane and the antral floor (not in the facial wall of the antrum). Numerous anatomic studies have confirmed this fact. It is to be borne in mind that the division of the alveolar nerves results in leaving the teeth

vital but insensitive. This frequently leads to loss of the teeth as there is no warning of the advance of tooth decay.

During the surgical removal of upper dead teeth pathologic perforations through the antral floor are frequently encountered. If necrosis is present the dead bone is removed. When the antral membrane is normal, thin and moving with respiration, it is not disturbed. A thick inflammatory membrane is rigid and should be incised so that the antral cavity may be examined for granulation tissue and polypi.

Removal of bone from below the antral membrane must be thorough as it is most important to leave the wound clean and free of any operative débris such as crumbs of bone and shreds of abscess wall; this will insure a smooth, uneventful repair, free from postoperative suppuration. Gauze packs are not allowed to remain in the wound, but are used only to control bleeding and to prevent bone chips from falling into the sinus. The gums are sutured with interrupted dermol which is removed on the fourth day. The alveolar process may be trimmed sufficiently to permit a flap to cover the antral opening. Uniformly good results have been obtained by this technique.

In my student days it was customary to irrigate a sinus for many months. Today I seldom find it necessary following surgery unless there is a postoperative rise in temperature. Several of my patients developed influenza shortly after operation and the postoperative fever induced me to irrigate the sinus which was found clean.

If, however, irrigation should be indicated some days after operation a No. 9 American soft rubber catheter is introduced through the healing incision, the patient's head is lowered to between the knees and 2 liters of a warm ½ per cent solution of saline with boric acid added are run through by gravity. It may be advisable to do a preliminary shrinking of the nasal ostium. When the irrigation is finished the suction may be applied to the catheter end to dry the cavity completely.

SUMMARY

Metastasis with serious sequelae, from dental focal infection manifesting slight clinical symptoms, is an established fact.

In the acute diffuse infection, operative interference should be most conservative, limited to establishing free drainage until the process is localized. Moist heat applications have been found valuable in maintaining circulation of the blood, inflammation is defensive, when circulation becomes blocked the defense fails.

Careful surgical dissection under direct vision in a field unobscured by blood and the removal of all operative débris is essential to success.

A SIMPLE METHOD FOR DETERMINING THE COAGULATION TIME OF THE BLOOD

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THE coagulation time of the blood is of tremendous importance in operative procedure. There are records of severe hemorrhage and even death due to the failure of the surgeon to protect the patient and himself.

There are many methods described for determining the coagulation time of the blood. All are more or less accurate, some require unusual skill and nearly all require apparatus or solutions, or both. In hospital practice, the surgeon depends largely upon the laboratory to make his tests, but minor operations are often done in the home or office and there the test must be made by the physician.

The following inexpensive and simple method may thus prove useful. It has been in use in the Wheatley-Provident Hospital for five years without the occurrence of a single fatality due to the inaccurate determination of the coagulation time. The finger is cleansed and punctured in the usual manner. The first drop of blood is discarded because of likely contamination. The second is allowed to remain for two minutes. Then the corner of a piece of absorbent paper is placed at the center and the blood from this part absorbed. (Fig. 1.) If coagulation has started, a ring (Fig. 2) will be seen at the periphery. The blood will coagulate there more quickly and remain adherent to the skin. If no ring is seen another drop is allowed to remain for four minutes and the procedure repeated. If still there is no ring, it is again repeated and the period increased until coagulation time is determined and safety of operative procedure established.

FIG. 1.

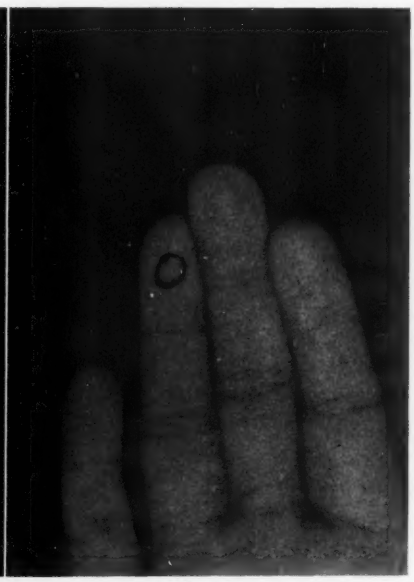

FIG. 2.

This method is sufficiently accurate, and is inexpensive and simple in operation.

CASE REPORTS

INTESTINAL OBSTRUCTION ASSOCIATED WITH ANOMALIES OF ROTATION AND FIXATION OF THE INTESTINE: REPORT OF A CASE

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IMPORTANT developmental changes which consist of an increase in length and in rotation and fixation are required to transform the primitive embryonic digestive tube into the adult digestive system. This paper is concerned with anomalies of fixation. However, without increases in length, rotation and fixation would not be necessary. Ideally, the process of fixation should take place only after increase in length and after the completion of rotation. Ideals are rarely attained during development. Actually, in practice, one encounters a great many variations of fixation of the intestine, but most of them are within what may be considered the limits of the normal. Anomalous fixation may be associated with failure of the first, second or third stages of intestinal rotation.[1] The lack of fixation associated with failure of the second or third stages of intestinal rotation or occurring in the presence of a normally rotated intestine may be the cause of intestinal obstruction. The intestinal obstruction is produced by an abnormal mobility which permits twisting of a segment or the whole intestinal mass. It is the object of this report to record an extraordinary instance of recurring intestinal obstruction which was due to volvulus of the intestinal mass. A failure of the second and third stages of intestinal rotation with lack of subsequent fixation was found at the time of surgical operation. (Fig. 1.)

REPORT OF CASE

A man, aged 24 years, came to The Mayo Clinic complaining of intermittent attacks of abdominal cramps which had occurred since birth. During the first three months of life he had had great difficulty in retaining food and in gaining weight. The attending physician had seriously considered the possibility of congenital pyloric stenosis. After the patient had become three or four months of age he had begun to gain weight and strength and subsequently had attained normal development in spite of frequent attacks of abdominal pain. The attacks of abdominal pain always had occurred very suddenly. The pain had usually been referred to the lower part of the abdomen and had been intense and cramp-like. In all the attacks much borborygmus, abdominal distention and incessant vomiting had been present. The attacks had lasted from a few hours to two days or until he had begun to pass flatus. He had discovered early in life that an attack might be completely relieved by taking a "full enema" (about 2,000 c.c. of fluid). In the interim between attacks he had been comfortable, his appetite had been good and his bowel motions had been regular without the use of laxatives. His appendix had been removed during one of the attacks. At the time of the appendectomy it had been observed that the cecum was situated in the left half of the abdomen. A second operation had been performed for "adhesions."

Physical examination revealed that the patient was well developed but somewhat undernourished. He was 5 feet and 10 inches (177.8 cm.) in height and weighed 130 pounds (59 kg.).

Roentgenologic examination revealed that the colon was situated mainly on the left side of the abdomen. The cecum was in close approximation to the sigmoid colon and descending colon. Physical examination did not disclose any other abnormality.

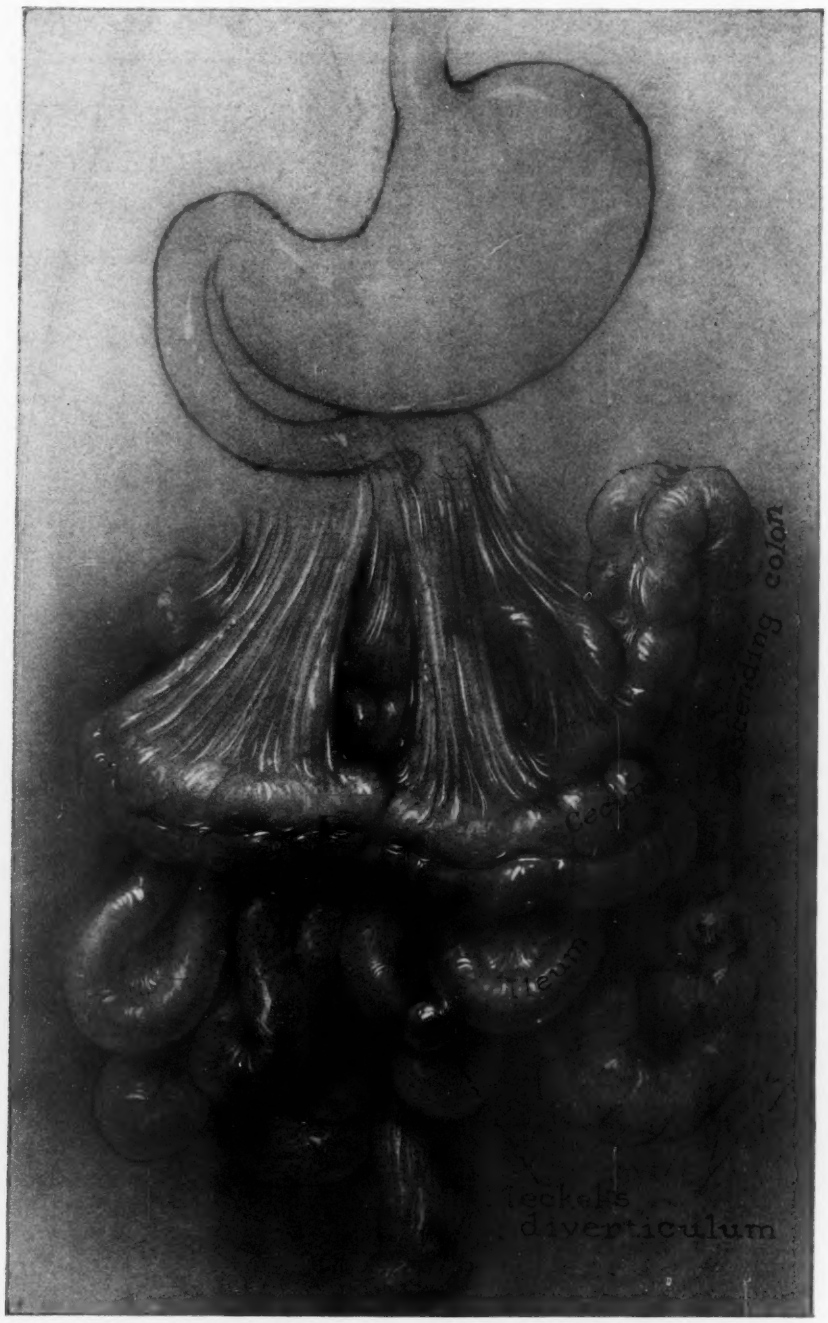

FIG. 1. Schematic representation of intestine and colon as observed at time of surgical operation.

At operation the total mass of the intestine was found suspended from a mesentery which was attached in the upper part of the abdomen in the general region of the ligament of Treitz. The base of the mesenteric attachment extended a short way down the left side of the abdomen. The mesentery was thick and heavy and loops of the small intestine appeared to be rolled within its leaves. In some sections of the coiled and rolled mesentery, which contained loops of the small intestine, it was difficult to determine whether or not herniation of loops

of the small intestine had occurred. On retracting the mass of the intestine forward and out of the abdomen, it was observed that there were no posterior attachments of any part of the intestine to the right of the midline of the abdomen. The lack of attachment of the small intestine permitted its free rotation in an arc of 180 degrees in either a clockwise or counterclockwise direction. The cecum and sigmoid and the transverse segments of the colon were in close approximation and were loosely adherent to the small intestine. The cecum had not rotated; it appeared to be rolled posteriorly and was attached to the sigmoid segment of the colon and to a portion of the descending colon.

A Meckel's diverticulum was present about 15 inches (37.5 cm.) proximal to the cecum. The diverticulum was about 3 or 4 cm. in diameter and was shaped like the neck of a bottle. It was not removed because it had a wide non-obstructing neck and also because of the danger of prolonging the operation. The superior mesenteric vein was unusually large, about 2.5 cm. in diameter in its proximal segment, and was very prominent. The superior mesenteric artery also was prominent. The appendix had been removed at a previous operation.

The mesentery, which suspended the cecum, ascending and transverse segments of the colon, was freed from its attachment, which was situated in the region of the ligament of Treitz. The cecum and the ascending segment of the colon were carried across and fixed with silk sutures to the lateral and anterior portions of the abdominal wall in approximately their normal position. The omentum of the transverse colon was brought across to cover the small intestine.

During the first seven or eight days after the operation the patient was nauseated and unable to retain food. He then began to eat, and to gain weight and strength, and he seemed to be normal in every respect. A roentgenologic examination of the colon one month after operation revealed that the cecum and ascending colon had remained where they had been fixed at the time of the operation. On dismissal the patient said that he felt better than he had at any time.

COMMENT

The lack of fixation of the mesentery may be compatible with digestive health, but when symptoms do occur they may be the result of volvulus, intussusception or occasionally, internal hernias. The commonest type of absence of fixation of the mesentery occurs when there is failure of the third stage of intestinal rotation. In cases in which there is a failure of the third stage of rotation, the cecum and proximal segment of the colon are left with varying degrees of mobility. It is necessary to emphasize that in our experience the so-called cecum mobile does not usually produce symptoms. We have found it a rather common occurrence.

Keyes recently reported two instances of failure of mesenteric fixation during the third stage of intestinal rotation in 148 cadavers. In neither had any known abdominal symptoms occurred during life.

Anomalous fixation of the mesentery which accompanies failure of the first or second stages of intestinal rotation and which is capable of producing serious symptoms may be divided into two groups: (1) the group in which there is failure of fixation of the small intestine, which makes possible the occurrence of volvulus of the entire mass of small intestine about the mesenteric pedicle,[2] such as occurred in the case reported in this paper; and (2) the group in which there are persistent congenital mesenteric bands which cause an intermittent and at times complete obstruction.[4] Gardner and Hart have reported two cases in which the anomaly belonged to the first group, and they reviewed the ninety-eight cases that had been reported in the literature. In eighty-eight of the ninety-eight recorded cases, volvulus resulted from twisting of the mass of the small intestine about the mesenteric pedicle. In the remaining ten cases the anomaly was the result of what these observers designated as "reverse rotation," which caused obstruction in the transverse colon. We have studied and recorded our experience in fifteen cases in which intermittent intestinal obstruction was caused by persistent congenital mesenteric bands accompanied by failure of the second stage of intestinal rotation.

In conclusion we wish to reiterate that anomalies of intestinal rotation and fixation of the mesentery are commonly observed. These anomalies are not usually productive of symptoms.

REFERENCES

1. Frazer, J. E., and Robbins, R. H. On factors concerned in causing rotation of the intestine in man. *J. Anat. & Physiol.*, 50: 75–110 (Oct.) 1915.
2. Gardner, C. E., Jr., and Hart, Deryl. Anomalies of intestinal rotation as a cause of intestinal obstruction: report of two personal observations; review of 103 reported cases. *Arch. Surg.*, 29: 942–981 (Dec.) 1934.
3. Keyes, E. L. Anomalous fixation of the mesentery: report of two cases. *Arch. Surg.*, 38: 99–106 (Jan.) 1939.
4. Wakefield, E. G., and Mayo, C. W. Intestinal obstruction produced by mesenteric bands in association with failure of intestinal rotation. *Arch. Surg.*, 33: 47–67 (July) 1936.

The anterior pituitary appears to be necessary for the absorption of glucose at a normal rate from the digestive tract. . . . The "diabetogenic" hormone of the anterior pituitary probably prevents the prodigal waste of important, but sometimes small, carbohydrate reserves which, for example, are maintained in spite of fasting.

From—"The Physiology and Pharmacology of the Pituitary Body," vol. II, by H. B. Van Dyke (University of Chicago Press).

CYSTADENOMA OF THE PANCREAS

REMOVAL THROUGH VERTICAL INCISION IN THE GASTROHEPATIC OMENTUM

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CYSTADENOMATA of the pancreas are of interest to the surgeon scientifically, because of their rarity and insufficiently studied pathology and they are of considerable practical importance to him so far as the technique for their successful removal is concerned. With this in mind, the case history of this patient is recorded, for further critical review.

Mrs. B. G., a woman of nervous constitution approaching the age of 44, for two years complained of a darting pain in the right lower abdominal quadrant, which radiated into the right groin. The pain was never severe. It was thought referable to the appendix by the patient but there were no characteristic seizures or findings. Occasionally there was also some pain in the right sacral region.

The patient readily became tired. Her eating habits and appetite were generally good, but she felt restrained in eating because of the pain in the right lower quadrant which followed. Her weight before operation was about 150 pounds.

There was habitual constipation, more pronounced in the previous one or two years. No complaints were referred to the urinary bladder. A hard mass in the abdomen had been noted a few days before.

The menses, originally normal, during several years past were prolonged to five or six days and small clots appeared. There was, however, no menstrual trailing. Beyond some bearable permenstrual pain no pronounced molimina occurred. The last menstrual period had occurred two weeks previously at the estimated time.

She had had two normal deliveries, one at the age of 15, the other at 25. No abortions, curettements or operations were reported.

Twelve years before the patient had met with an automobile accident in which her shoulder was bruised, but apparently no sequel remained except headache. She was subject to influenzal infection during the winter season, particularly two years before. There was a maternal history of thyroid disorder.

The mid-abdomen was prominent at the site of the mass, which was intra-abdominal, well circumscribed and apparently close to the abdominal wall. In relation to the umbilicus it could be outlined as follows: 2½ inches above, 3 inches to the left, 2½ inches to the right and 1½ inches below. (Fig. 1.) When it was held fixed under the palpating hand no traction appeared to be exerted by respiration. The mass gave the impression of limited mobility towards the left, but this proved to be erroneous when the abdominal wall was relaxed under anesthesia; its actual origin from the upper abdomen and fixed attachment posteriorly were then unmistakable. At the previous clinical examination attempts to shift the mass caused slight pain. Old circumumbilical striae were noticed in the abdominal wall, but there was no change in the umbilicus, no infiltration, no enlarged veins.

The upper border of the liver was ⅔ inch below ensiform level and its lower border at "intersection point." The gall-bladder area was not tender, the lower pole of the right kidney not palpable, the epigastrium above the abdominal mass was flat and soft. The descending colon could be palpated readily and was impacted but not tender. The cecum was empty and there was no tenderness over the right beta point. No mass was palpable in the hypogastrium. Pelvic examination revealed nothing beyond a slight dextroversion of the uterus with a mild catarrhal endotrachelitis.

The urine was free of albumin and sugar.

The general impression was of a pedunculated myoma of the uterus. Carcinoma of the

transverse colon was considered at first, but under anesthesia the likely diagnosis rested between an omental mass and cyst of the pancreas.

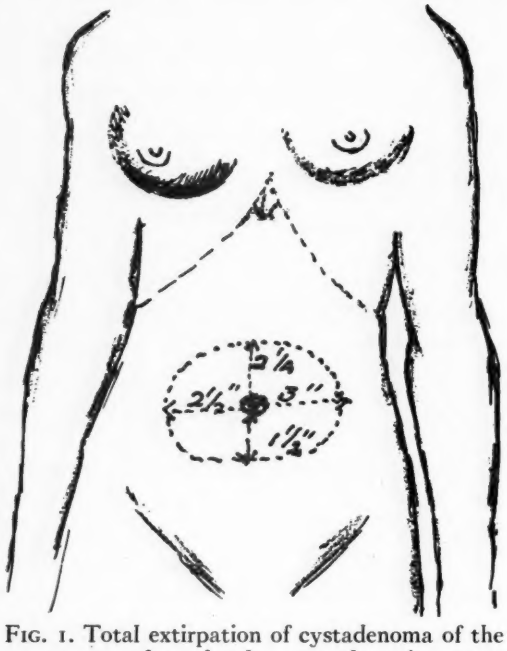

FIG. 1. Total extirpation of cystadenoma of the pancreas through the gastrohepatic route. Impression of tumor location and size by palpation and percussion before anesthesia.

Operation was done May 6, 1937 and lasted one hour forty-five minutes. Gas-oxygen-ether anesthesia was preceded by morphine sulfate gr. ¼ and atropine sulfate gr. $\frac{1}{150}$. Near the end of the operation intravenous infusion of physiologic saline solution was administered: 400 c.c. were given in the operating room and an additional 200 c.c. in bed.

The real origin of the abdominal tumor then became more evident; the mass appeared fixed and had shifted towards the upper abdomen. The incision was placed slightly extramedian, being carried to the left of the umbilicus, and after exploration through the smaller incision, it was extended upward to the ensiform notch.

The stomach, pylorus and first part of the duodenum appeared normal. The liver margin was high. The table rack was raised at the level of the upper abdomen to put the patient into the gall-bladder posture. The transverse colon showed no lesions. There was no omental tumor or mass.

When the stomach was drawn downward, a bluish surface with grape-like contour could be seen through the gastrohepatic omentum. Connection of the mass with the pancreas was verified by palpating through the foramen of Winslow with the left index finger. (Fig. 2.)

Instead of dividing the attachment of the

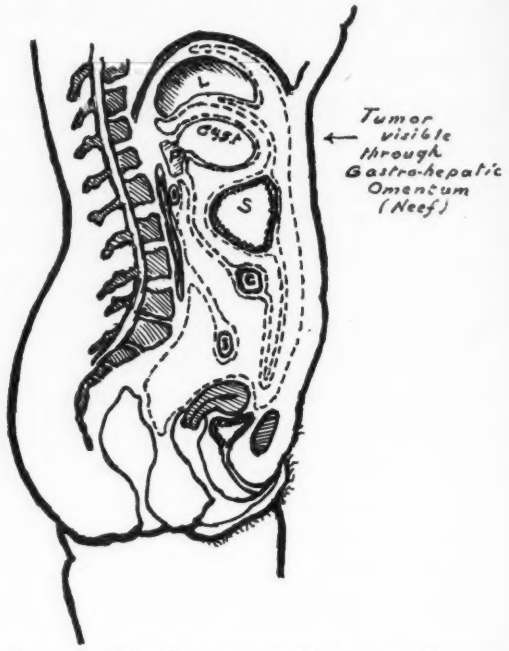

FIG. 2. Complete removal of cystadenoma through vertical incision in the gastrohepatic omentum. No drainage. Primary healing.

lesser omentum to the lesser curvature of the stomach, we made a vertical slit in the gastrohepatic omentum to gain access to the lesser sac and the space behind the stomach. Through this route the tumor was liberated and removed by stripping and dissection, with careful adherence to the surface of the mass and palpation for larger vessels to be secured. As the tumor was gradually lifted from the surface of the pancreas posteriorly and towards the left, a vein the size of a goosequill and a pulsating artery of similar caliber were identified. The vein was ligated and a small portion of pancreatic tissue removed with the growth. Some time was consumed in obtaining thorough hemostasis and a dry tumor bed. (Fig. 3.) No drain was used. The slit in the gastrohepatic ligament was not closed by suture, but coapted well when the stomach was allowed to return to its normal site. The cystic tumor was removed intact with the exception of one small loculus which was punctured during the procedure and from which spurted a clear fluid, 4 to 6 inches high from a pin point opening. (Fig. 4.)

During and after operation the pulse rate reached a maximum of 140, but its quality

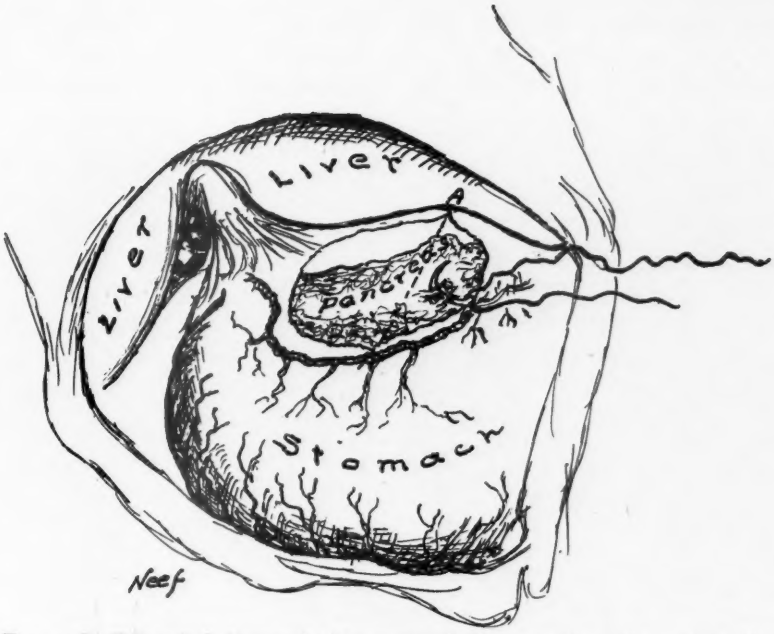

Fig. 3. Pedicle stitch inserted after removal of tumor through vertical slit in lesser omentum.

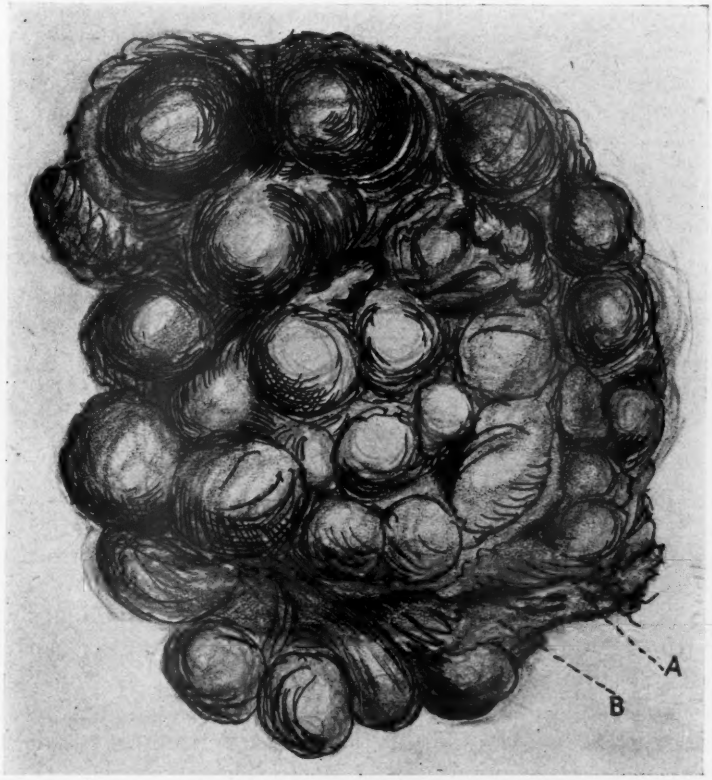

Fig. 4. Reconstructed diagram of mass. Tumor measurements 9 × 9 × 6 cm. Individual cysts from 0.4 to 2.4 cm. in size. A, strip of pancreas. B, site of origin near tail of pancreas.

remained relatively good until the final step in tumor removal. Then it became poorer but was promptly restored as the warm saline took

attached to the surface. Section revealed a multilocular cystic structure throughout. The individual cysts were smooth-walled and con-

FIG. 5.

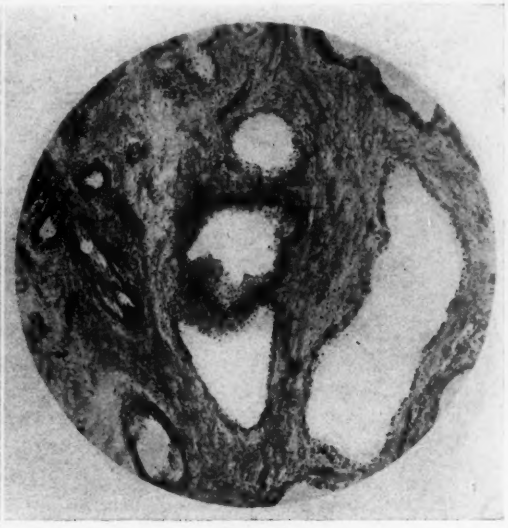

FIG. 6.

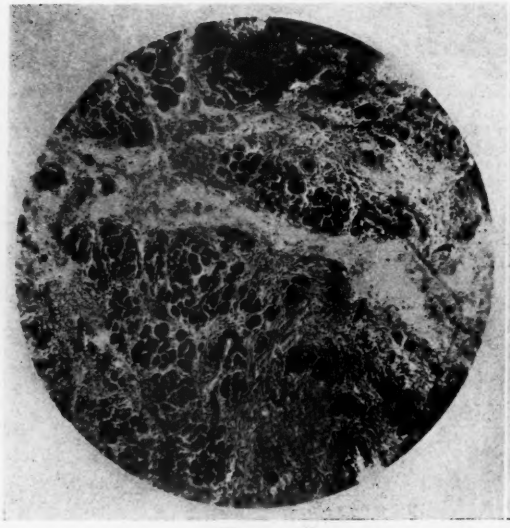

FIG. 7.

FIG. 8.

FIGS. 5 TO 8. Photomicrographs of cystadenoma of pancreas.

effect. At the end of operation the pulse rate was 140; the pulse quality was good, eyelids tonic, pupils narrow, color slightly flushed, breathing quiet. Within an hour after the return to bed the pulse rate dropped to 116 and remained satisfactory.

The mass measured 9 by 9 by 6 cm., and its surface was studded with multiple thin-walled cystic nodular projections ranging in size from 0.4 to 2.4 cm. in diameter. A small piece of pinkish and finely lobulated pancreatic tissue approximately 2 by 1 by 0.6 cm. was firmly tained serous fluid brownish-yellow or, more often, dark brown in color. (Fig. 4.)

Microscopic examination showed congeries of cysts of various sizes *lined with one or multiple layers of small undifferentiated cells.* The outlines of some of these were poorly delineated, while others were sharply defined, the latter being cuboidal or polyhedral in shape. The nuclei were small, spheroidal and rather chromatic, the cytoplasm apparently non-granular and usually clear. The latter was evidently free of lipoid material since no fat was

demonstrable by the Scharlach R stain. There were no definite papillary ingrowths, though in localized areas the cells were heaped up in the *tumor, though there was a quantity of pancreatic tissue attached to the outer surface.* The growth was well vascularized and in some regions

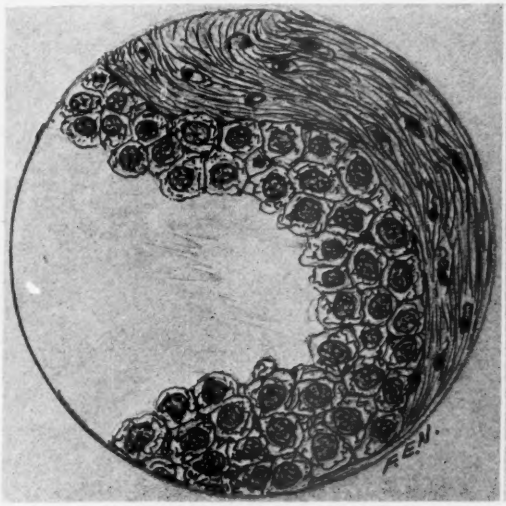

Fig. 9. Multiple cell layer in wall of cyst. (× 333.)

Fig. 10. Cysts lined with a single layer of epithelium.

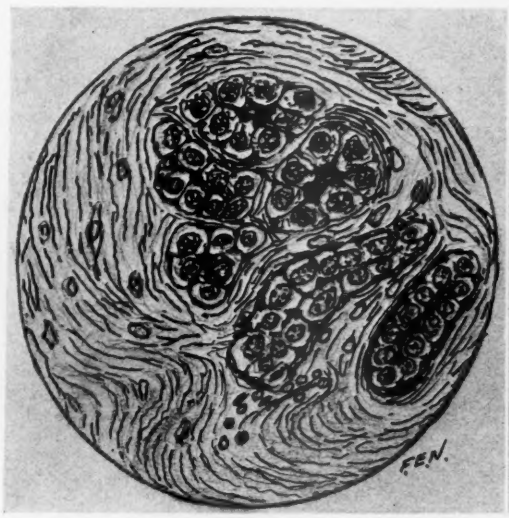

Fig. 11. Section of pancreas. (× 333.)

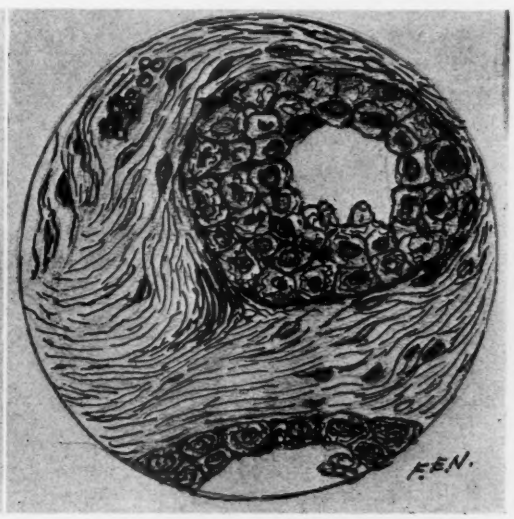

Fig. 12. Section of a small cyst under high power.

form of blunt or slender processes which projected into the cyst cavities. These cavities were generally empty, but some contained serum, erythrocytes and a few desquamated or inflammatory cells. The supporting septa differed in thickness and consisted of connective tissue of varying density and cellularity, loose and edematous in some places, dense or hyaline in others. In some of the septa there were a few glandular spaces lined with cells similar to those lining the cysts and there were also small nests of cells reminiscent of islet cells. *Neither pancreatic acini nor ducts could be identified in the* showed scattered hemorrhages together with an irregular inflammatory infiltration, the inflammatory cells consisting chiefly of small round cells. (Figs. 5–14.)

A review of the various slides obtained from this tumor indicated that about 85 per cent of the cyst spaces were lined with a single layer of epithelium; in about 3 per cent the epithelium was from two to five cells deep, making due allowance for the plane of section, and in approximately 1 per cent small localized papillae were observed. This behavior is quite

analogous to that observed in multilocular cysts of the ovary.

Postoperative Course. Hypodermoclysis with saline was instituted towards evening of the day of operation and 1,000 c.c. fluid were given in this way. Within eighteen hours after operation the maximum rectal temperature reached 103.4°F., with respiration 18, pulse 120 soft, fair in volume, and regular. The chief complaint was thirst and this was met by starting the rectal drip with plain water at the rate of 16 drops per minute continuously, except in the morning and evening from 6 to 8 o'clock. Morphine sulfate gr. $\frac{1}{4}$ with atropine sulfate gr. $\frac{1}{150}$ were administered as needed.

No liquid by mouth was tolerated for forty-eight hours after operation. The period of thirst was followed by one of heartburn on the third day. The patient was still thirsty and quite irritable. Bed exercises for the extremities were encouraged and the patient was tilted to the right or to the left side. The heartburn was finally overcome by feeding thin cream of wheat with milk, cream soups and lime water made palatable with milk and given in 2 ounce doses.

On the fourth day gas distress and gas pain were relieved by means of the siphon enema with plain water and without the help of pituitrin. On the fifth day the gas pain waned and the patient was put on a convalescent's diet.

The sixth day following operation towards evening the patient was greatly upset because of general abdominal and epigastric distress. The temperature rose to 102°F., the pulse varying from 108 to 116, of good quality. No heartburn was present. The next morning, that is six and a half days postoperative, the menses appeared. The first dressing was done at this time and all the stitches were removed. The incision line was dry and there was no infiltration or local disturbance.

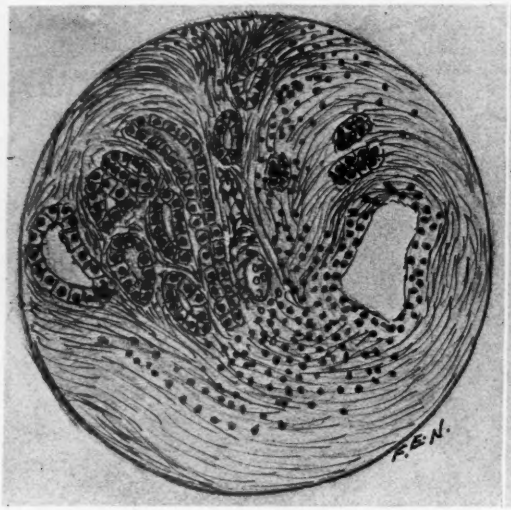

FIG. 13. Intercalated tubules and round-cell infiltration in connective tissue stroma.

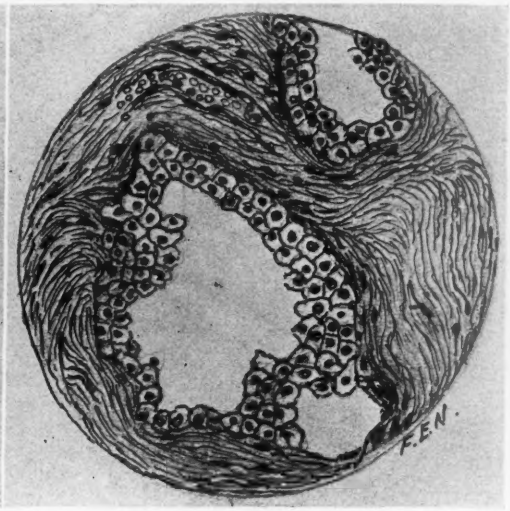

FIG. 14. Cyst sections. (× 125.)

On the eighth day postoperative the temperature reached its maximum of 102.2°F. The cytologic and chemical blood examination at this juncture showed the following:

Blood Cytology

Hemoglobin		68 per cent (10.6 Gm.)
R.B.C.		4,980,000
W.B.C.		29,800
Differential	Polys.	84 per cent (22 per cent immature cells)
	Lymph.	14 per cent
	Mono.	2 per cent

Blood Chemistry

Urea N	9.3
Creatinine	0.5
Uric acid	2.5
Sugar	97.
CO_2 combining power	55.7 volumes per cent

The urine contained no acetone, diacetic acid or sugar.

Thereafter the temperature dropped gradually and was again normal on the thirteenth day after operation. Seventeen days after operation the blood cytology was as follows:

Hemoglobin		62 per cent (9 Gm.—standard 15.5 Gm.)
R.B.C.		3,100,000
W.B.C.		5,800
Differential	Polys.	64 per cent (12 per cent immature)
	Lymph.	30 per cent
	Mono.	4 per cent
	Eos.	2 per cent

The patient was allowed out of bed and went home two days later, after a total hospital stay of nineteen days.

CLINICAL RETROSPECT

1. There is an apparent disparity with reference to location and mobility as ascertained before anesthesia and during it. As the diagram of the office findings in the recumbent posture showed, the mass presented itself in the umbilical region and conveyed the impression that it moved with respiration. Under anesthesia, on the other hand, it was most prominent in the epigastric area and appeared to be more fixed; nevertheless positive differentiation between an omental mass and pancreatic cyst could not be made before operation. On exploration, however, a bluish mass nearly 4 inches (about 9 cm.) in diameter could be seen between the liver and lesser curvature of the stomach, pushing the gastrohepatic omentum before it. That it was a tumor arising from the pancreas was easy to verify by introducing the left index finger into the foramen of Winslow.

2. In this approach, deviating from the usual, the lesser omentum was incised vertically to avoid division of blood vessels supplying the lesser curvature. Through this opening the multilocular tumor together with a small strip of pancreas was extirpated.

3. Drainage was omitted. This would seem to lessen postoperative intra-abdominal irritation due to the presence of a foreign body in the territory of the solar plexus. It also allows the divided gastrohepatic omentum to coapt perfectly and heal without interference. Besides obtaining a minimal degree of intra-abdominal cicatrization and adhesion, the union of the abdominal wall itself is interrupted at no point so that there remains no postoperative sinus when the sutures are removed at the first dressing—in this instance six and a half days after operation.

One year after operation the patient's average gross weight was about 150 pounds. She had lost weight before undergoing operation. The weight one month after operation was 133 pounds; in a further three weeks 134½; six months postoperative 144½, and 140½ on revision one year after operation. At this time she excluded sweets from her diet.

Some symptoms remained which were referable to occasional hyperthyroidism with palpitation on the heart. The patient mentioned that she still had some abdominal pain in the right side, which, however, was indefinite and absent for two weeks at a time. Her appetite was very good, food no longer upset her digestive function, there was no discomfort after eating. She also stated that she had not been constipated for nine months after her operation, slept well, and experienced no pain in the region of the scar.

SUMMARY

It is noteworthy that the history of this patient contained very little that was suggestive of her real disorder.

Initially even pelvic origin of the tumor was considered, because it projected itself largely in the umbilical area as the diagrams which were made at this time indicate. A neoplasm of the transverse colon or an omental mass might also be found in this location. Not until the patient was under anesthesia was its attachment in the upper abdomen clearly defined making a cyst of the pancreas likely.

The gastrohepatic omentum was found stretched over the presenting tumor and was incised vertically instead of according to the usual technique. This opening, with slight lateral traction, afforded sufficient working room. Vertical incision had the advantage of being in itself bloodless and obviated the necessity of severing the vascular attachment of the gastrohepatic

omentum to the lesser curvature of the stomach.

As was expected the removal of a small strip of pancreas at the vascular base of the tumor had no noticeable effect on the patient's blood chemistry.

Another deviation from the usual procedure was the avoidance of all drainage of the tumor bed. The reflex upset caused during convalescence by drains lodged in this region, as well as the formation of adhesions and delay in the complete closure of the abdominal wound may be factors of some importance; and, where there is reason to believe that the hemostasis is secure, the bed is dry and the wound is clean such drainage appears unnecessary.

A review of the literature reveals that a number of conditions which are not true neoplasms have been classified as pancreatic cysts, and, in other instances the pathology was too meager to establish that the tumor was a true cystadenoma. The growth which is described here is histologically a benign neoplasm and belongs to the group of true cystadenomata.

The number of cystadenomata of the pancreas recorded as successfully removed is comparatively limited. In this patient it was an advantage that the cystic tumor had its vascular attachment to the tapering body of the pancreas. If it had arisen nearer the head of the gland, the simpler technique might not have been applicable.

REFERENCES*

1. Adler, H. M. Zwei Fälle von Pankreascyste. *Virchows Arch. f. path. Anat.*, Suppl., 177: 154, 1904.
2. Alexandrow. Zur Kasuistik der Totalextirpation der Pankreascysten. *Zentralbl. f. Chir.*, 35: 319, 1908.
3. Baldwin, J. F. Cystic tumors of the pancreas. *Ohio State M. J.*, 4: 547, 1908.
4. Battle, W. H. Pancreatic cyst possessing undue mobility. *Med. Press & Circ.*, 33: 595, 1907.
5. Berkman, J. M., and Bannick, E. G. Huge multilocular cyst of pancreas; 2 cases. *Proc. Staff Meet., Mayo Clin.*, 8: 185, 1933.
6. Bessel-Hagen. Zur operativen Behandlung der Pankreascysten. *Verhandl. d. Deutsch. Gesel. f. Chir.*, 29 Kongress 1900. Also, *Arch. f. klin. Chir.*, 62: 157, 1900.
7. Bevan, A. D. Pancreatic cyst. *S. Clin. North America*, 3: 887, 1923.
8. Bozeman, N. Removal of cyst of pancreas weighing twenty and one-half pounds. *M. Rec.*, 21: 46, 1882.
9. Brackel, A. Zur Kenntniss der Pankreascysten. *Deutsche Ztschr. f. Chir.*, 49: 293, 1898.
10. Bull. Report of a case of pancreatic cyst. *New York J. Med.*, 46: 376, 1887.
11. Burmeister. Über Luftpumpen. Drainage bei Pankreascysten. *Anat. u. klin. Chir.*, 75: 183, 1905.
12. Busse. Gallensteine und Pankreascyste. *Deutsche med. Wchnschr.*, 477, 1911.
13. Carter, R. F., and Slattery, L. Cystadenomata of the pancreas. Surgical report of a case. *Am. J. Digest. Dis. & Nutrition*, 3: 705, 1936.
14. Carwardine, T. Pancreatic cyst in a boy aged seven. *Bristol Med.-Chir.*, 26: 319, 1908.
15. Connolly, D. L. Note on a case of pancreatic cyst in a child 14 months. *Lancet*, 1: 803, 1911.
16. Culler, R. M. Cure of pancreatic fistula by the roentgen ray. *J. A. M. A.*, 75: 20, 1920.
17. Cushing, H. W. Traumatic rupture of the pancreas. Formation of hemorrhagic cyst: operation, followed by pancreatic fistula and recovery. *Boston M. & S. J.*, 138: 429, 1898.
18. De Francisco, G. Un caso di doppia cisti del pancreas. Estirpazione total etc. *Rif. med.*, 28: 206, 1912.
19. Delagénière (Le Mans). Des kystes glandulaires du pancréas. Une observation. *Arch. prov. de chir.*, 9: 209, 1900. Also, *Arch. prov. de chir.*, 15: 193, 1906.
20. Eade. Double pancreatic cyst. *New Zealand M. J.*, 6: 15, 1907.
21. Edling. Zur Kenntniss der Cystadenome des Pankreas. *Virchows Arch. f. path. Anat.*, 32: 110, 1905.
22. Eha, C. E. Case of congenital pancreatic cyst. *J. A. M. A.*, 78: 1294, 1922.
23. Einhorn, M. On pancreatic cysts. *Am. J. M. Sc.*, 169: 389, 1925.
24. Esau. Pankreascyste als Ursache einer Stenose der Flex. Coli sin. *Wien. klin. Wchnschr.*, 47, 1907.
25. Exner, A. Zur Kasuistik und Therapie der Pankreascysten. *Wien. klin. Wchnschr.*, 18: 803, 293, 1905.
26. Fiessenger, N. and Cattan, R. Cystadenoma of head of the pancreas. *Bull. et mém. Soc. méd. d. hôp. de Paris*, 53: 497, 1929.
27. Fitz, R. H. Multilocular cystoma of the pancreas. *Am. J. M. Sc.*, 120: 184, 1900. Also, *Tr. A. A. Physicians*, 15: 255, 1900.
28. Friedenwald, J., and Cullen, T. S. Seven cases of pancreatic cysts. *Tr. A. A. Physicians*, 41: 317–342, 1926. Also, *Am. J. M. Sc.*, 172: 313, 1926.

* With few exceptions the references to the literature are restricted to cystadenoma of the pancreas. Of the group of reported cases assembled in this list by Dr. S. A. Ritter only a very small number was subjected to radical surgical treatment with successful extirpation of the pancreatic growth.

29. Gibbon. Clinical Report of a case of cyst of the pancreas and interstitial pancreatitis. *Charlotte M. J.*, 5: 69, 1906.
30. Göbell, R. Über die Totalextirpation von Pankreascysten. *Verbandl. d. deutsch. Gesell. f. Chir.*, 36: 361, 1907.
31. Gravemann. Ein Beitrag zur Lehre der Pankreascysten. Diss. Kiel, 1902.
32. Gussenbauer, Carl. Zur operativen Behandlung der Pankreascysten. *Arch. f. klin. Chir.*, 29: 355, 1883.
33. Gutierrez, A. Implantation into the stomach of a pancreatic fistula following cyst. *Internat. Abstr. Surg.*, 43: 214, 1926.
34. Gwathamy, L. Pancreatic cysts and report of a case. *Tr. South. Surg. & Gynec. A.*, 25: 52, 1912.
35. Hadley. Multilocular cyst of the pancreas. *Brit. M. J.*, 1: 1112, 1914.
36. Hamilton, C. S. Prolonged and profuse postoperative drainage of pancreatic cyst and use of radium. *Surg., Gynec. & Obst.*, 35: 655, 1922.
37. Harms, C. Cystadenoma of pancreas; case. *Zentralbl. f. allg. Path. u. path. Anat.*, 55: 53–56, 1932.
38. Harsha, W. M. Pancreatic cyst. *Ann. Surg.*, 39: 455, 1904.
39. Hartman, M. Kystes du pancréas. *Rev. de chir.*, 11: 409, 1891.
40. Heiberg, K. A. Die Krankheiten des Pankreas. Wiesbaden, 1914. J. F. Bergmann.
41. Henricius. Über die Cysten und Pankreascysten und über ihre chirurgische Behandlung. *Arch. f. klin. Chir.*, 54: 388–470, 1897.
42. Hersche, J. Operation einer Pankreascyste von seltener anastomischer Lagerung. *Wien. klin. Wchnschr.*, 5: 727, 1892.
43. Hinz. Beitrage zur Pankreaschirurgie. *Deutsch. med. Wchnschr.*, 9: 399, 1912.
44. Hirst, B. C., and Veeder, B. S. A case of pancreatic cyst. *Proc. Path. Soc., Phila.*, 13: 128, 1910.
45. Honigmann. Zur Kenntniss d. Pankreascysten. *Deutsch. Ztschr. f. Chir.*, 80: 19, 1905.
46. Huefer, W. C. Cystadenoma of pancreas. *Arch. Surg.*, 5: 261, 1928.
47. Huerre. Analyse d'un liquide provenant d'un Kyste pancréatique. *J. de Pharm. et de Chim. de Paris*, 27: 287, 1908.
48. Jaboulay. Kyste du pancréas. *Prov. méd.*, 15: 354, 1901.
49. Janes, R. M. Cystadenoma of pancreas, 2 cases. *Brit. J. Surg.*, 23: 809–815, 1936.
50. Jelezareff. Kystoma Dermoidale Kolossale pancreatis. *Medizinski Napredak*, 1901.
51. Judd, E. S. Cysts of the pancreas. *Minnesota Med.*, 4: 75, 1921.
52. Judd, E. S., Mattson, Hamlin, and Mahorner, H. R. Pancreatic cysts; report of 47 cases. *Arch. Surg.*, 22: 838, 1931.
53. Jurasz, A. Zur Frage der operativen Behandlung der Pankreascysten. *Arch. f. klin. Chir.*, 164: 272, 1931.
54. Karpeles, M. J. Pancreatic cyst; report of case. *Am. Med.*, 9: 782, 1905.
55. Keitler, H. Zur Kasuistik der Pancreascysten. *Wien. klin. Wchnschr.*, 12: 764, 1899.
56. Kerr, A. A. Cysts and pseudocysts of the pancreas with report of cases. *Surg., Gynec. & Obst.*, 27: 40, 1918.
57. Kleinschmidt. Beitrage zur pathologischen Anatomie und Klinik der Cystadenome des Pankreas. *Med.-Natur. Arch.*, 1: 177, 1907.
58. Kopfstein, J. Cyste pancreatica. *Casop. lek. Česk.*, 14: 427, 1905.
59. Korp. Treatment of cysts and pseudocysts of the pancreas. *Med. Press & Circ.*, 91: 512, 1911.
60. Körte, W. Die chirurgischen Krankheiten und die Verletzungen des Pankreas. *Deutsche Chir.*, 234, 1898. Also, Zur Behandlung der Pankreascysten und Pseudocysten. *Deutsche med. Wchnschr.*, 30: 536, 1911.
61. Krauss. Uber Pankreascysten. *Zentralbl. f. Bakteriol.*, 55: 8, 1910.
62. Kuhn, H. P., Schutz, C. B., and Helweg, F. C. Cystadenoma of pancreas; case. *J. Missouri M. A.*, 27: 570–572, 1930.
63. Kurpeles, M. J. Pancreatic cysts; report of case. *Bristol Med.-Chir. J.*, 26: 319, 1908.
64. Lagautte. Kyste du pancréas. *Lyon méd.*, 114: 1164, 1910.
65. Lazarus, Paul. Beitrag zur Pathologie und Therapie der Pankreaserkrankungen mit besonderer Berucksiehtigung der Cysten und Steine. *Ztschr. f. klin. Med.*, 51: 95, 203; 52: 146, 381, 1904.
66. Le Monut. Cas de kyste pancréatique. *Arch. gén. de chir.*, 8: 11, 1912.
67. Lepoutre, C., and Busser, F. Cystadenoma of pancreas. Bull. *Assoc. franç. p. l'étude du cancer*, 22: 215, 1933.
68. Lynbenetski and Fofanoff. Two cases of pancreatic cyst. *Russk. Vrach.*, 5: 359, 1906.
69. Maes, Urban. Cysts of the pancreas with the report of one case. *Internat. Clin.*, 2: 95, 1931.
70. Malan, A. Tumore cistico del pancreas. *Policlino*, 16: 178, 1909.
71. Malcolm, J. D. A case of complete removal of a multilocular cystic tumor of the pancreas. *Lancet*, 1: 1676, 1906.
72. Marks, S. B. Pancreatic cyst. *Kentucky M. J.*, 11: 551, 1912.
73. Martens. Zur Chirurgie der Pankreascysten. *Deutsche Ztschr. f. Chir.*, 100: 306, 1909.
74. Martin, A. Ein Fall von Pankreascyste. *Virchows Arch. f. path. Anat.*, 120: 230, 1890.
75. Mathieu. Les Kystes du pancréas (vrai Kystes et pseudokystes). *Paris-méd.*, 11: 23, 1911.
76. Mayo, W. J. Pancreatic cyst. *M. Rec.*, 45: 168, 1894.
77. McBurney, Charles. Cyst of the pancreas. *Ann. Surg.*, 19: 492, 1894. Also, *Arch. Surg.*, 11: 619, 1925.
78. McCann, F. J. A large pancreatic cyst simulating an ovarian tumor. *Proc. Roy. Soc. Med.*, 4: 235, 1912–1913.
79. McKechnie, R. E., Jr., and Priestley, J. T. Multilocular polycystic tumor of pancreas. *Canad. J. M. & S.*, 36: 592, 1937.
80. McWhorter, G. L. Cysts of the pancreas. *Arch. Surg.*, 11: 619, 1925.
81. McWilliams, C. Recurrent pancreatic cyst. *Ann. Surg.*, 54: 434, 1912.

82. McWilliams, C. A. True pancreatic cyst. *S. Clin. North America*, 3: 439, 1923.
83. Moniet. Quoted by Mauclaire. Kyste du pancréas. *Semaine méd.*, 32: 33, 1912.
84. Moschcowitz. Cyst of the tail of the pancreas. *Ann. Surg.*, 58: 563, 1913.
85. Münzer, Max. Pankreascysten. *Zentralbl. f. d. Grenzgeb. d. Med. u. Chir.*, 6: 490, 573, 664, 1903.
86. Murray, F. W. Cyst of the pancreas, 3 cases. *Ann. Surg.*, 53: 558, 1911.
87. Neuffer, H. Surgical therapy of pancreatic cysts. *Arch. f. klin. Chir.*, 170: 488, 1932.
88. Noodenbos, W. Extirpation of cystadenoma of pancreas. *Nederl. tijdschr. v. verlosk. en gynaec.*, 2: 5546, 1929.
89. Ochsner. Pancreatic cyst. *Ann. Surg.*, 43: 949, 1906. Also, *Arch. f. klin. Chir.*, 39: 446, 1889.
90. Opie. Diseases of the Pancreas. 1910, 266.
91. Oser. Diseases of the pancreas. Nothnagel Encycl. of Practical Med., 1905.
92. Papadopaulos, H. Un cas de kyste du pancréas; troubles digestifs résultant de la compression du colon transverse. *Grèce med. Syra.*, 7: 29, 1905.
93. Payr, Edwin. Pankreascyste, seltener Topographie, Operation und Heilung. *Wien. klin. Wchnschr.*, 11: 629, 1898.
94. Pels-Leusden. Beitrag zur Pankreaschirurgie. *Charité-Ann.*, 26: 148, 1902.
95. Pinault. Kyste du pancreas. *Bull. méd. de Quebec*, 8: 262, 1906.
96. Pollord, B. Three cases of cyst of the pancreas etc. *Brit. M. J.*, 1: 594, 1899.
97. Potherat. A propos Kyste du pancreas. *Bull. et mém. Soc. de chir. de Paris*, 37: 911, 1911.
98. Preisich. Cyste der Bauchspeicheldrüse. Jahrbuch der Kinderheilkunde, 1901, 53.
99. Primrose, A. Pancreatic cysts and pseudocysts; report of a case of total extirpation by an extraperitoneal method. *Surg., Gynec. & Obst.*, 34: 431, 1922.
100. Railton, T. C. A case of pancreatic cyst in an infant. *Brit. M. J.*, 2: 1318, 1896.
101. Reeke, T. Cystadenoma of pancreas; case. *Frankfurt. Ztschr. f. Path.*, 40: 444, 1930.
102. Riedel. Ein Fall von Pankreascyste. *Arch. f. klin. Chir.*, 32: 994, 1885.
103. Robson and Cammidge. The pancreas, its surgery and pathology. Phila. and London., 1907. W. B. Saunders Co.
104. Roić, V. Zur Extirpation der Pankreascysten. *Wien. klin. Wchnschr.*, 27: 294, 1914.
105. Roman. Zur Kasuistik der Pankreastumoren. *Virchows Arch. f. Path.*, 209: 234, 1912.
106. Santy, P., Mallet-Guy, P., and Croizat, P. Tumeur polykystique au corps du pancréas. Pancréatectomie gouche. *Lyon chir., Bull. Soc. de chir. de Lyon*, 33: 689, 1936.
107. Schlesinger. Ein Beitrag zur Bewertung der Rontgen Untersuchung bei der Diagnose von Pankreascysten. *Med. Klin.*, 1027, 1912.
108. Schlönvogt, E. Cystadenoma of pancreas. *Nowotwory*, 6: 145–158, 1931.
109. Schmidt, W. Ein Fall von total Extirpation einer Pankreascyste. *München. med. Wchnschr.*, 54: 2480, 1907.
110. Schwartz. Kystes et tumeurs kystiques du pancréas. *Bull. et mém. Soc. de chir. de Paris*, 37: 945, 1911.
111. Scotti, G. Adeno-cistoma papillefero del pancreas. *Arch. per le sc. med.*, 1906. Abstr., *Zentralbl. f. allg. Path. u. path. Anat.*, 18: 844, 1907.
112. Seefisch. Mitteilung über Pankreascysten. Aus Chirurgischen Abteilung des Krankenhauses am Friedrichskain. *Deutsch. Ztschr. f. Chir.*, 59: 153, 1901.
113. Senn, Nicholas. Surgical treatment of cysts of the pancreas. *Am. J. M. Sc.*, 90: 17, 1885. Also, Surgery of the pancreas as based on experiments and clinical researches. *Am. J. M. Sc.*, 92: 141, 1886; 93: 121, 1887. Experimental Surgery, Chicago, 1889. W. T. Keener.
114. Sitzenfrey. Zur Extirpation der Pankreascysten. *Ztschr. f. Geburtsh. u. Gynäk.*, 54: 98, 1905.
115. Sofrano. Adeno-cistoma papillifero del pancreas. *Arch. per le sc. med. Torino.*, 30: 184, 1906.
116. Speese, J. J. Cystadenoma of the pancreas with extension to the abdominal wall 10 years after drainage of a pancreatic cyst. *Ann. Surg.*, 61: 759, 1915.
117. Starck. Zwei Fälle cysticher Pankreasgeschwülste. *Beitr. z. klin. Chir.*, 29: 713, 1901.
118. Steinmann. Pankreascyste. *Korrespondenzbl. f. Schweiz. Ärzte*, 17: 608, 1909.
119. Takayasu. Beitrag zur Chirurgie des Pankreas. *Mitt. a. d. Grenzgeb. d. Med. u. Chir.*, 3: 89, 1898.
120. Tavernari, A. Papilliferrus cystadenoma of pancreas; case. *Patbologica*, 23: 207, 1931.
121. Telling and Dobson. Infant, aged 12 months, large pancreatic cyst drained successfully. *Brit. J. Child. Dis.*, 6: 202, 1909.
122. Treamaine, W. S. Cyst of pancreas successfully removed. *Tr. Am. Surg A.*, 6: 557, 1888.
123. Vecchi, G. Internal drainage of a large cyst by means of pancreatic-gastrostomy. *Wien. klin. Wchnschr.*, 48: 45, 1935.
124. Walker, H. Pancreatic cyst, rupture, recovery. *Brit. M. J.*, 2: 360, 1911.
125. Willis, A. M., and Budd, S. W. Pancreatic cysts with report of a case. *Surg., Gynec. and Obst.*, 20: 688, 1915.
126. Wolff, E. Pankreascysten und Pseudocysten. *Beitr. z. klin. Chir.*, 74: 487, 1911. Also *Munchen. med. Wchnschr.*, 572: 2443, 1910.
127. Wyss, A. A. Beitrag zur Kentniss der cystischen Pankreastumoren. Diss., Kiel, 1904. Diss., Basel, 1904.
128. Yanooskiy, S. M. Cystadenoma; case. *Vestnik. khir.*, 45: 83, 1936.
129. Young, E. L. Jr. Multilocular cystadenoma. *New England J. Med.*, 216: 334, 1937.
130. Zeller. Extirpation einer Pankreascyste. Mediz. Korresponenbl. des Württenberg. *Arztl. Landesvereins.*, 14, 1910.
131. Zweiful, P. Extirpation einer Pankreascyste. Heilung der Kranken. *Zentralbl. f. Gynec.*, 18: 641, 1894.

SACROCOCCYGEAL TERATOMATA IN THE NEWBORN

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SACROCOCCYGEAL tumors are composed of many different types of tissue; there is found in them fetal nerve tissue with ganglion cells, remains of the central canal, choroid plexus, epidermis, smooth and striated muscle, bronchial segments, intestines, liver, suprarenals, fibrous connective tissue, mucosa, cartilage and bone. Congenital postsacral teratomas usually contain a preponderance of nerve tissue, so that rests of the neural layer have been considered as the source of these tumors.

The tumor is usually a benign growth. The degree of cell differentiation is important in the consideration of malignancy, as the more the cells approach the adult type, as in the formation of glands and of organs, the less is the possibility of malignancy. Gross clinical evidence of adult tissue is indicated by muscle twitchings or peristalsis in the tumor or the demonstration of bone tissue. Teratomas found anterior to the sacrum and posterior to the rectum do become malignant.

Stewart, Alter and Craig reported a tumor which was anterior to the sacrum, in a boy of 2 years and 11 months, which metastasized to the liver, lungs and regional lymph nodes. Parin reported six cases undergoing malignant degeneration with metastases, and deVeer and Browder reported one case. Renner and Goodsit also reported a case of malignant teratoma anterior to the sacrum following the removal of a postsacral teratoma.

These tumors vary greatly in size. They may be anterior to the sacrum and entirely intrapelvic, or they may present a huge external mass as large as the infant's head externally and dorsal to the sacrum and coccyx. They are found most frequently in females. Galbert estimates that they occur once in 34,582 births; Ewing states that one-third of the fetuses with sacrococcygeal teratoid tumors are born dead, while 90 per cent of the others die soon after birth.

These tumors contain both cystic and solid elements, and may increase in size very rapidly in the first months of life. This rapid increase in size, according to Hansmann and Berne, is not to be taken as a sign of malignancy but as a growth of the tumor parallelling the infant's growth.

The following is a report of teratoma in a newborn, with complete removal of the tumor on the fourth day of life, in which recovery followed.

The baby was admitted to the Springfield City Hospital one hour after birth. The mother, aged 20, was a primipara, in whose family twins were common. She had noticed during her pregnancy a large mass in the upper right zone of the abdomen. Labor was slow; after the head and shoulders were delivered all further progress ceased and traction proved futile. On palpation an obstructing mass was discovered. The infant was finally delivered by introducing the hand along the posterior wall of the vagina over the baby's buttocks and flexing the baby over the mother's abdomen.

The infant was normal except for a solid and cystic tumor mass attached to the sacrum, measuring about 15 cm. in diameter. This mass extended laterally under the gluteal muscles almost to the trochanters. The skin over the mass was greatly thinned; the capillaries in the skin were injected in the upper portion of the tumor and this area was dark red in color. No pulsations or change in size were noted when the child's position was changed. The tumor extended farther on the right side than on the left, and over this area there were constant muscular twitchings. The lower extremities were normal and reacted to sensory stimuli.

The blood count was normal: red cells 2,700,000; hemoglobin 58; leucocytes 7,000 and polymorphonuclears 32 per cent.

X-ray examination with a catheter in the rectum showed no communication between the rectum and the tumor, although the mass

FIG. 1. Section showing some of tissues in bronchi. Note cartilage, pseudostratified ciliated "respiratory" epithelium and mucus glands. (× 250.)

appeared to be adherent to the posterior rectal wall. Many small dense masses were visualized within the tumor.

Due to the fact that the skin over the vertex of the mass showed signs of breaking down, operation was performed on the fourth day of life. An oval incision was made about the base of the tumor. The skin was easily separated from many dark colored cysts which made up the upper part of the mass. The lower solid portion of the tumor was dissected from the rectum and, with some fibers of the gluteus maximus muscles, was removed. There was very little bleeding, but the child was shocked. Transfusion was done on the table. The wound healed without infection.

The specimen weighed 502 gm. and after partial removal of fluid from the tumor, a decrease of weight of 182 gm. was noticed. Three different tumors were present, the largest the size of a large grapefruit of distinct nodular structure. The covering skin measured 12 by 4 cm. The skin was closely adherent to the tumor and was not movable over the mass.

Several different masses, containing nodes of similar structure were observed. Many smaller noduli and many cysts contained water-clear

FIG. 2. Area of teratoma showing mixture of types of epithelium and connective tissue. Small island of hyalin cartilage, stratified ciliated columnar epithelium, tall columnar epithelium containing mucus and small mucous glands resembling those seen in bronchi. There are numerous smooth muscle fibers in a loose connective tissue stroma. There are numerous dilated lymph spaces. (× 75.)

and yellowish-white turbid fluids. After removal of the fluid the tumors presented a honeycomb appearance. Other nodes of different gross appearance showed cartilage, connective tissue and a substance which resembled brain tissue.

Microscopic examination showed several different types of tissue. Bronchial structures, such as cartilage, pseudostratified ciliated "respiratory" epithelium, and mucus glands, were noted; brain, adipose and fibrous connective tissues with striated muscle fibers were also found. The predominant material present was brain tissue with smooth muscle fibers running through it. Brain surrounded by heavy deposits of melanin was found in some sections. One area showed choroid plexus with columnar epithelium lining a small cyst. Many mixed types of epithelium and connective tissue were also present. Rindfleisch has aptly called these tumors a *bistologic pot-pourri.*

According to H. W. Hundling a number of developmental errors account for these tumors. "It is in this area that the caudal

termination of the primitive streak should most accurately attain its evolution and involution, the neurenteric canal develop and disappear, the anus complete the intestinal tube, the posterior fissure properly close, the coccyx and sacrum develop, and the inferior extremities adapt themselves to the trunk. During the third month of fetal life the neural tube extends to the end of the vertebral canal and into the tail bud. The spinal canal, developing faster than the soft parts, draws along the neural tube adherent to it. The coccygeal portion of the neural tube is bent into a loop of which the deeply situated limb is attached to the posterior surface of the coccyx, while the superficial one assumes a more dorsal position. This deep limb disappears at four months and the superficial one continues to develop. The spinal chorda and the mesoderm develop at the same rate. The tail bud is reduced with a reduction of the mesoderm. The spinal cord is now longer than the vertebral column."

After the third month the vertebral column is longer than the cord and the caudal end of the cord is drawn upward.

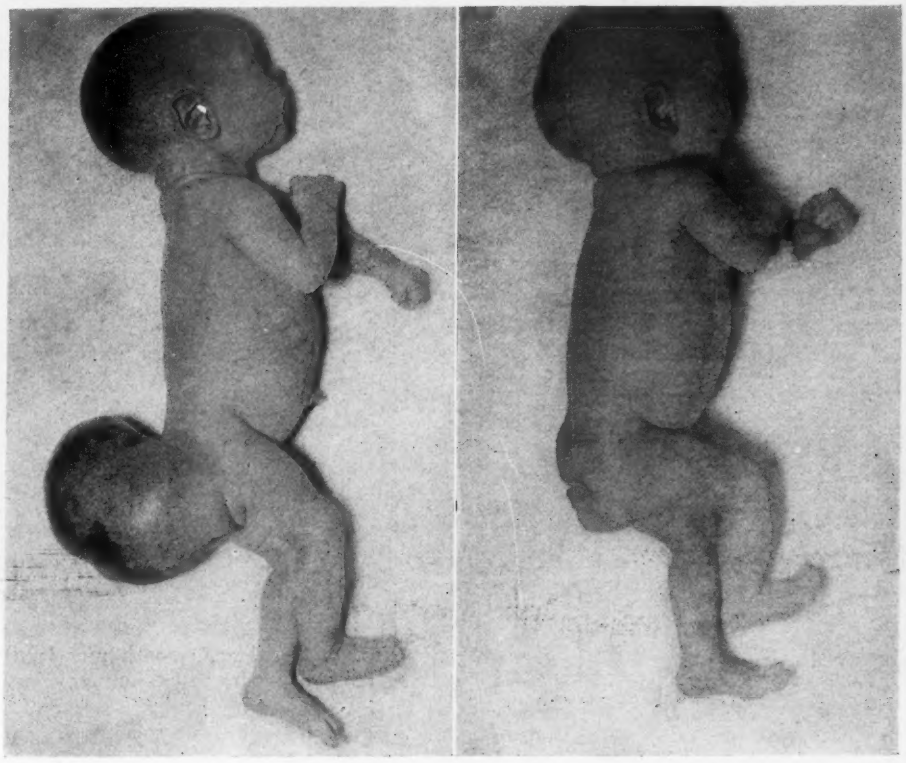

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Fig. 3. Photographs of subject.

"While the endoderm is forming the caudal intestine, the dorsal canal and dorsal cord, the mesoderm, the connective tissue, blood vessels, vertebra and muscles, and the ectoderm is forming the primitive streak, the medullary tube and its vestiges, there is a continuation between the central canal of the spinal cord and the primitive alimentary canal around the caudal extremity of the notocord known as the neurenteric canal. When the proctodeum or primitive anus, invaginates to form part of the cloacal chamber, it meets the gut anterior and above where the neurenteric canal opens into it, hence there is for a time a segment of intestine behind the anus termed the postanal gut."

From this complicated fetal development and recession of the different tissues many theories have arisen as to the origin of sacrococcygeal tumors. Their early

development is fairly definite proof of their relationship to embryologic processes. Hansmann and Berne think the embryo-

Fig. 4. Area of choroid plexus. Note single layer of cuboidal cells and preponderance of nervous tissue elements. (× 375.)

logic structures occurring in this region, in the hindgut, proctodeal membrane, and in the neurenteric canal, are sufficient to account for the varying types of tissue in these tumors. According to Ewing, although some of the tumors may with considerable certainty be referred to a single embryonal structure, the majority are more complex and probably involve more than one of these embryonal remnants and some additional anomalies of development.

Tumors of the lumbosacral region, according to Middledorph, Ristchl and Nasse, are due to proliferation of the remains of the neurenteric canal, hindgut and medullary canal in association with ectodermal and mesodermal inclusions. Middledorph was the first to ascribe their origin to the postanal gut. Grosser, Lewis and McMurrick also think the neurenteric canal, the postanal gut or the neural canal is the source of these tumors.

Teratomata may possibly arise from parthenogenetically developing sex cells. This modification of the old bigerminal

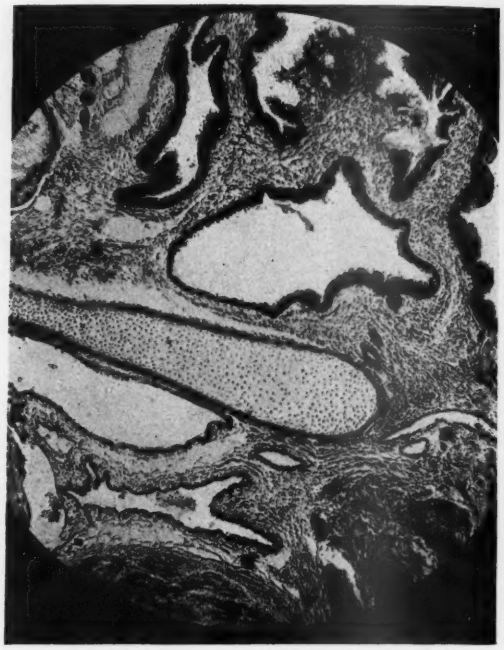

Fig. 5. Cartilage, smooth muscle, transitional epithelium and tall columnar epithelium containing mucin. Brain tissue is again seen in the stroma. (× 100.)

theory has been given much support by Bosaeus in his work on the origin of ovarian embryomata.

MacCallum believes that teratomatous tumors in the sacrococcygeal region, which approach the complexity of twin inclusions, are best explained as originating from an isolated blastomere with varying potentialities. He states: "A teratoma resulting from parthenogenetic development of a sex cell would be of the nature of an offspring, while one derived from an isolated blastomere would be of the same generation as the host, a twin." The histogenesis of these tumors is usually considered to be independent development of a blastomere during segmentation of the ovum or the development of a misplaced ovum. In view of the constant position of these tumors it is difficult to accept this explanation.

Ziegler states: "If a teratoma can be found to contain very diverse tissue formation which, at least in part, may be

identified as representing rudiments of organs which, however, are superfluous for the individual in whom they are found, we may consider such a tumor as a heterochthonous teratoma, or a bigerminal implantation that is a rudimentary twin. On the other hand, if the teratoma contains only diverse tissues and cysts whose production does not necessarily imply the existence of a second division, the tumor may be looked upon as an autochthonus teratoma or a nongerminal implantation."

The bigerminal implantation was held by Virchow, Ahlfeld and others, while Bonny and Bland-Sutton held to the theory of dichotomy of the fetal axis, or the suppressed fetus theory. Adami thought a growth of totipotential cells at the inferior growing point was the cause of teratomata, and Tourneux claimed their cause was a persistence the medullary coccygeal vestige.

Theory after theory has thus been presented as to the causation of these tumors and no one has been universally accepted. It is the consensus of opinion that due to the complicated embryologic development of the lower axis of the body, tumors of various types of tissue are possible, but it is impossible to attribute the development of sacrococcygeal teratomas to any one fetal structure.

The treatment is surgical removal. How soon after birth the tumor should be removed depends on the condition of the skin, whether the growth is extra- or intrapelvic, and the rate of growth. These tumors may ulcerate through the skin by pressure necrosis and become infected; if they are intrapelvic they may, by their rapid growth, cause intestinal obstruction.

CONCLUSIONS

1. A case is presented of sacrococcygeal teratoma in a newborn, with operation and recovery four days after birth.

2. Many kinds of tissue, including brain, cartilage, muscle, bronchial, and glandular tissue were found in this tumor.

3. Theories for the causation of these tumors have been reviewed.

4. The treatment recommended is immediate surgical intervention.

REFERENCES

Renner, R. R., and Goodsitt, E. *Am. J. Cancer*, 7: 35.

Hansmann, G. H. Congenital cystic tumor of neurenteric canal. *Surg., Gynec. & Obst.*, 42: 124, 1926.

Hundling, H. W. Ventral tumors of the sacrum. *Surg., Gynec. & Obst.*, 38: 518, 1924.

Hansmann, G. H., and Berne, C. J. Sacrococcygeal tumors. *Arch. Surg.*, 25: 1090, 1932.

Keen, W. W., and Copelin, W. M. L. Sacrococcygeal tumors. *Surg., Gynec. & Obst.*, 3: 661, 1906.

Stewart, J. D., Alter, N. M., and Craig, J. D. Sacrococcygeal teratomata with malignant degeneration in childhood. *Surg., Gynec. & Obst.*, 50: 85, 1930.

Weintraub, S., and Young, R. Sacrococcygeal tumor. *Arch. Pediat.*, 50: 472, 1933.

Utter, H. E., and Bates, R. C. Sacrococcygeal tumors. *Rhode Island M. J.*, 10: 36, 1927.

deVeer, J. A., and Browder, J. Sacrococcygeal teratoma. *Ann. Surg.*, 37: 408.

Kümmel, W. *Virchows Arch. f. path. Anat.*, 118: 37, 1889.

Mallory, F. B. *Am. J. M. Sc.*, 103: 263, 1892.

Duncan, H. A. Retrorectal tumor. *M. J. & Rec.*, 120: 108, 1924.

Irvine, G. M. Congenital sacral teratoma. *Indian M. Gaz.*, 65: 568, 1930.

Fuss, E. M. Carcinomatous degeneration of a teratoma of the coccygeal region. *Arch. f. Chir.*, 159: 515, 1930.

Gallety, A. Presacral tumors of congenital origin. *J. Obst. & Gynaec. Brit. Emp.*, 31: 226, 1924.

Pendalai, K. G., et al. Sacrococcygeal teratoma containing gliomatous, chordomatous and ependymal carcinomatous tissues. *J. Path. & Bact.*, 27: 139, 1924.

Sawday, A. E. Partial intestinal obstruction in a child with a post rectal teratoma. *Brit. M. J.*, 2: 685, 1925.

A CASE OF PERITONITIS COMPLICATED BY PROGRESSIVE MUSCULAR DYSTROPHY*

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THE symptomatology in this case of generalized peritonitis complicated by progressive muscular dystrophy is of particular interest in that the atrophy which involved both recti and other abdominal muscles, altered the clinical picture usually present in peritonitis. The points of interest were the absence of spasticity and rigidity which was accounted for at the time of operation and subsequent necropsy. This also explained the presence of the marked distention as a prominent symptom found at the time of examination.

C. B., male, a farm hand, aged 31, was admitted to the neurological ward in 1930. The following history was obtained at that time: At the age of 12, the patient had noticed wasting of the thenar muscles; the muscles of the arms and legs were normal. At the age of 17, he noticed marked wasting of the muscles of the upper extremities. Somewhat later, wasting started in the leg muscles and there was difficulty in walking. After 1924, a gradual but progressive development of muscular weakness occurred.

The patient was a 31 year old white male with wasted arm, scapular, trunk and thigh muscles. He went through complicated routine movements in getting into the sitting posture. His pupils reacted to light and accommodation; the fundi were negative. Conjunctival and corneal reflexes were present, as was the pharyngeal. Deep reflexes were absent in the upper extremities. The abdominals were present, more active on the right. The cremasteric reflex was present and active, the patellar absent, and the Achilles present. Other pathologic reflexes were not noted. No sensory disturbances were elicited. There was considerable atrophy of all the extremities, more marked in the upper. The characteristic winged scapulae and characteristic postures while attempting to rise were observed. Faradic responses were all present and speech was unaffected. The feet presented slight pes cavus.

The family history was not contributory as the patient did not remember his parents.

A juvenile type of progressive muscular dystrophy was diagnosed.

On April 24, 1938, the patient was again seen. He had had diffuse abdominal pain with distention and eructations of two days' duration. The onset had occurred three days prior to admission, about one and one-half hours following a meal. The following morning he took some milk of magnesia. This was followed by a good bowel movement and some relief of pain. Later in the day, and especially the following day, the abdominal pain and general distress became unbearable and the patient consented to be admitted to the surgical ward.

The patient, now 39, appeared acutely ill, with flushed and apprehensive face. The tongue was coated and dry. There was evident marked wasting of all the muscles of the extremities, chest and trunk. In order to allay pain, the patient lay quietly. His temperature was 103, pulse 95, respirations 20, blood pressure 115/65. Heart and lungs appeared normal. There was marked rebound tenderness and distention of the abdomen and also *complete absence of spasticity and rigidity*. The presence of peritonitis was apparent and immediate laparotomy was advised. However, the patient refused operation.

The following morning the tongue was heavily coated and moist. The abdomen revealed a lack of rigidity throughout (*muscular dystrophy*); tenderness was generalized and rebound tenderness marked. The urine was negative. The white count 14,600, polymorphonuclear leucocytes 83 per cent, lymphocytes 15 per cent, monocytes 2 per cent. Red cells numbered 3,200,000. Temperature was 102.4, pulse 100, respirations 22.

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The clinical picture was one of generalized peritonitis, distorted because of the presence of muscular dystrophy and the mentality of the patient. His apprehension bordered almost on panic and he had the mentality of a 10 year old. However, he finally gave his consent for an operation.

Under gas-oxygen-ether anesthesia a lower right rectus incision was made. All abdominal muscles showed fibrosis and atrophy. The rectus muscles were represented by a fibromuscular band. Free seropurulent fluid exuded under pressure from the general peritoneal cavity. The appendix was found ruptured and gangrenous, retrocecal, and bound down by a mass of adhesions. It was fragile and broke off in handling. The appendiceal stump was carbolized and purse-stringed. The terminal ileum appeared markedly inflamed and the ileocecal valve almost occluded. A cecostomy tube was purse-stringed through the anterior band of the cecum and the tube fed through the ileocecal valve into the ileum for a distance of about 8 inches. The cecum was sutured to parietal peritoneum and a Penrose drain was placed in the retrocecal fossa and pelvis.

The postoperative diagnosis was: ruptured appendix, with ileitis and general peritonitis.

On the first and second days after operation the patient's general condition was apparently good; distention was absent and the temperature was 101 degrees. On the third day marked distention appeared, with vomiting and abdominal tenderness. The cecostomy tube drained satisfactorily. Treatment for the peritonitis consisted of Wangensteen suction, sedation, and parenteral fluids. The general condition was slightly improved on the fourth day and continued fair for the following days. On the seventh day, the general condition appeared excellent—there was only slight distention and the cecostomy tube was draining. The eighth day showed a turn for the worse; greenish-black vomitus appeared, with moderate distention and a fecal discharge around the cecostomy tube. On the tenth day, the cecostomy tube sloughed out; profuse drainage of fecal matter occurred through the cecostomy wound and through the lower angle of the incisional wound. Marked distention and tenderness, especially on the right side of the abdomen, was noticed. From the eleventh to the eighteenth day, the temperature ranged from 101 to 103 degrees. The general condition showed a steady downward course. Consolidation in the left chest appeared on the eighteenth day, and on the ninteenth day the temperature was 105, the skin cold, clammy and cyanotic, the pulse thread-like, and labored respiration. Death followed.

Autopsy. The body, that of a young man about 39 years of age, was poorly developed and poorly nourished. There was a considerable amount of post-mortem hypostasis, particularly over the back, neck and abdomen. *There was no rigor mortis in any of the extremities.* Bedsores were found over the sacral region and over the right thigh. The body was delicate stature, with only a slight amount of hair; the hair over the pubes assumed female distribution.

The chest was flattened and its muscles markedly diminished in size and volume; the left anterior chest wall showed no pectoral muscle and the intercostals were atrophied. On the right side there remained a small amount of abdominal and chest muscle. Both lungs showed numerous adhesions to the chest wall as well as to the diaphragm. An extensive pneumonic process was present throughout the left lung with irregular yellowish patches suggestive of pyemic foci. The right lung showed only congestion, some pneumonic infiltration and pulmonary edema. A necrotic area containing liquefied dark material, obviously localized lung gangrene, had developed in the posterior axillary portion of the left lung.

The abdominal wound from below the umbillicus to the level of the inferior spine of the ilium had raw, ununited edges, its surface being gangrenous and covered with greenish exudate. The abdominal muscles were practically absent, so that the skin was the prominent layer of the abdominal wall. A large amount of pus was present in the peritoneal cavity, bounded by the right diaphragm and omentum. The small intestines were dilated, the loops of the ileum adherent to each other as well as to the abdominal wall and the right side of the abdomen. In the region of the cecum, loops of small intestines were matted together, encircling a pocket of pus; these loops were also adherent to the pelvic wall. One portion of ileum was necrotic. The cecum and ascending colon were involved in a suppurative process, the colon adherent to the duodenum, gallbladder and liver, while the cecum was entirely gangrenous and adherent to the abdominal wall. A large amount of pus, particularly

inspissated, was found in the region of the cecum. The abdominal wall covering the region was entirely necrotic. The distal half of the transverse and descending colon were contracted and apparently not markedly involved in the gangrenous process. A few adhesions were found between the upper sigmoid and pelvic wall, while a large pocket of pus was present in the left pelvic fossa. On the posterior abdominal wall there were numerous greenish masses.

The spleen, enlarged to twice normal size, was of soft consistency, hemorrhagic and bilobar, weighing 200 Gm. The left adrenal was decreased in size and appeared hemorrhagic on section. The right adrenal was somewhat decreased in size but otherwise showed no marked abnormalities. The pancreas was of usual size and, on section, appeared degenerated. The liver was larger than normal, its diaphragmatic surface showing an area about 10 cm. in diameter covered with yellow exudate, due to the accumulation of pus between the diaphragm and the dome of the liver. The gall-bladder was distended and covered by omental adhesions due to the infectious process in the peritoneum. It contained thick bile but no stones.

The muscles of the extremities appeared markedly atrophic, particularly those of the upper extremities. The muscles of the hand were all decreased in volume.

The pathological diagnosis was: (1) muscular dystrophy (facioscapulohumeral type); (2) acute gangrenous appendicitis; (3) acute peritonitis; (4) sepsis; (5) acute inflammation of ileum and cecum; (6) lobular pneumonia and pyemic abscesses of lung; (7) acute parenchymatous degeneration of internal organs.

SUMMARY

The absence of abdominal musculature due to progressive muscular dystrophy disguised the symptomatology in this case of ruptured appendicitis with peritonitis. The lack of spasticity and rigidity was accounted for by the absence of the abdominal musculature. The possibilities of symptoms being masked in other neurological diseases complicated by the acute abdomen, must be kept in mind.

ANGIOBLASTIC SARCOMA OF LIP*

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CARCINOMA of the lip is of relatively frequent occurrence, but sarcoma is a rare form of malignancy to affect this organ. Tibor De Cholnoky,[1] in 1934, reported four cases of sarcoma of the lip and from the literature gleaned only twenty cases described, quoted or mentioned, the records of all but five of which were incomplete from either a clinical or a pathologic standpoint. Most of the patients were senescent, although one was a child 1½ years old. The stated duration of the lesion varied from a few weeks to several months. There was a history of trauma in only one case.

The lesions varied from a few millimeters to several centimeters in diameter, were reddish-blue or deep red in color, bled easily and had a smooth or more or less lobulated surface which proceeded to ulceration. Histologically the lesions were classified as spindle-cell, round-cell and mixed-cell sarcomas, lymphangiosarcoma, neurogenic sarcoma and melanosarcoma; spindle-cell types predominated. Metastases occurred in two cases.

Wide surgical excision of the primary lesion was the predominant mode of treatment. Irradiation was less effective. Two patients died within a few months from generalized sarcomatosis. Cholnoky's cases were symptom-free six, six and eight months, and four years, respectively, after operation. In three of these four cases dissection of regional lymph nodes accompanied wide local excision of the lesion, while the lesion occurring on the upper lip was removed by electro-coagulation.

Cholnoky stated that there were two cases of sarcoma of the lip reported from the Göttingen Pathologic Institute among 1,248 cases of sarcoma of all anatomic locations. Sarcoma of the lip was not mentioned in Simmons's[2] series of 177 cases of sarcoma of the soft parts, in the review of tumors of connective tissue by Geschickter and Lewis[3] or in that of tumors of blood vessels by Geschickter and Keasbey.[4] Freilich and Coe,[5] in 1936, reported a case of angiosarcoma of the scapula and found twenty-nine cases of angiosarcoma recorded in the literature since 1918; reports of twenty-six of these were available; one of the twenty-six, that of M. Prati (1931) was primary in the lip; two were primary in the tongue. In the York Hospital from 1932 to 1935 there were 9,110 adult admissions (1,740 of these were maternity cases); in this period there was only one case of sarcoma of the lip—the one herein reported.

CASE REPORT

T. F., male, white, 53 years of age, was admitted June 16, 1935 with the complaint of a bleeding and painful lower lip. In April, he had been struck on the lower lip by a small stone while at work. The lip became swollen and a "blood blister" formed. The area did not heal but gradually enlarged and began to slough and bleed slightly. About four weeks before admission to the hospital the growth became painful to move, and sometimes it bled profusely. The family history was irrelevant. He was not a pipe smoker, nor had he ever been troubled with any ulceration, crack or sore on the lip prior to the trauma.

The physical examination revealed nothing remarkable, except the lesion of the lip. This covered an area about the size of a quarter dollar on the vermillion border of the lower lip, to the left of the midline, and protruded outward for at least ½ inch. It was of a lobulated

* From the Departments of Surgery and Pathology, York Hospital.

fungoid type, with a comparatively constricted base. The surface was fairly smooth, darker red at various places, and oozed blood. The superficial tissues were somewhat necrotic and sloughing. The regional lymph nodes were not palpably involved. There was a secondary anemia of moderate degree. The Kahn test was negative.

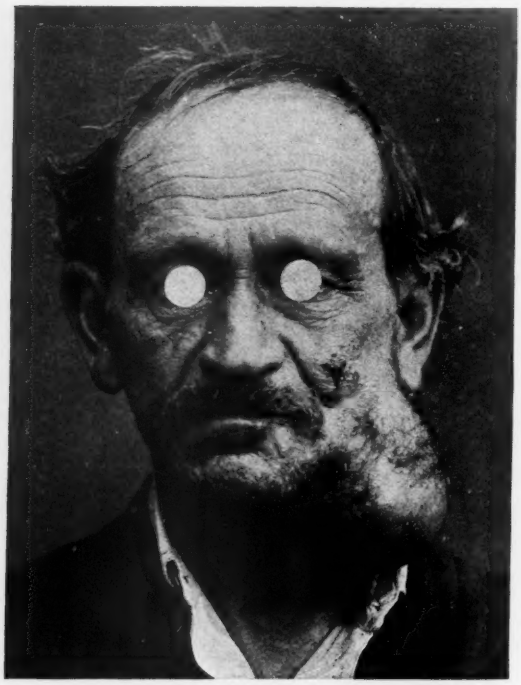

FIG. 1. Appearance of patient after resection of lip. A large metastatic tumor of the neck, of approximately four months' duration, is seen. No recurrence at operative site.

Removal of the growth and cheiloplasty were performed on June 17, 1935. A V-shaped incision was made in the lower lip, allowing approximately 2 cm. of lip tissue on either side of the growth. The left arm of the V began close to the left commissure, meeting the right arm of the V well over the mental region. These incisions sloped slightly from the skin toward the mucous membrane of the buccal surface, thereby giving greater thickness and insuring better healing and less thinning of the scar. Blair's lip clamps were applied on either side in order to control bleeding without excessive vessel ligation. The left commissure was then incised horizontally and a Bernard triangle was dissected from the upper lip, following the nasolabial crease and utilizing the mucous membrane of the back of the triangle for the vermilion border. In performing the cheiloplasty the skin sutures were placed first and superficially, but on the gingival side the sutures were placed deeply and made to include the fibers of the orbicularis oris, as in the repair of an anterior alveolar cleft. Oral hygiene was instituted before the operation and continued after the operation, including a sterile diet for the first forty-eight hours.

The tissue removed at operation was received in the laboratory in a solution of formaldehyde. It consisted of a wedge-shaped portion of lip, 2.5 by 2.0 by 1.5 cm., to the mucocutaneous margin of which was attached a pedunculated, fungoid, reddish-brown mass, not unlike a mushroom in size and shape, 2.5 cm. in diameter and 1.5 cm. thick. The outer surface was lobulated and ulcerated, largely covered with coagulated blood. Except for this coagulated blood the cut surface revealed a relatively homogeneous, solid, gray structure, poorly demarcated from the underlying tissues of the lip, at the ill-defined junction with which hemorrhage into the tissue was seen. The diameter of this junction was 1 cm., constituting a potential pedicle without appreciable length, the periphery of the mass overhanging and resting on the cutaneous and mucous surfaces of the lip.

Microscopically a rather densely cellular neoplasm composed of a matrix of pleomorphic cells supporting numerous vascular channels was seen. These vascular channels in sections were devoid of contents and for the most part did not have a definite wall, except that imparted by the bordering pleomorphic cells. These cells were fusiform, round, ovoid and polyhedral in shape. Those irregularly of fusiform shape predominated; some had a narrow elongated process; an occasional cell of stellate shape was seen. Cytoplasm and cellular processes everywhere were acidophilic. Nuclear variation in size, shape and chromatin content was a conspicuous feature. Many nuclei were strikingly hyperchromatic. The nuclei of the more differentiated cells were ovoid in shape, moderately pale and had an evenly distributed finely divided chromatin network. Nucleoli nowhere were prominent. There were a large number of mitotic figures.

The diffuse arrangement of the neoplastic cells, their lack of any definite grouping, the predominance of those irregularly of fusiform shape with nuclei having obscure nucleoli were features characteristic of sarcoma and not

characteristic of carcinoma. The inclusion of numerous apparently vascular channels, lined by neoplastic cells, indicated an origin from

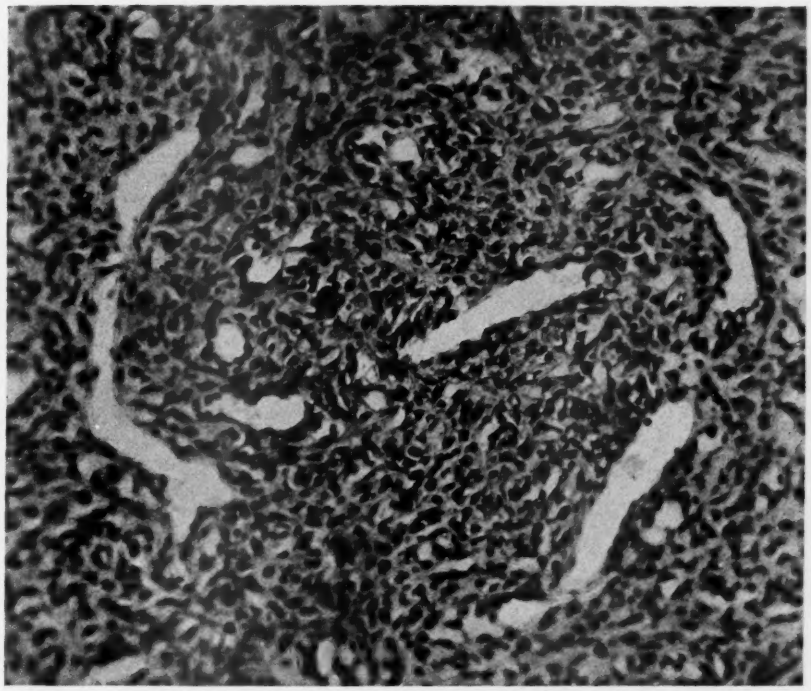

Fig. 2. Low power photomicrograph of tumor of lip. The diffuse heterogeneous arrangement of the neoplastic cells, among which ramify vascular channels, is seen.

vessel walls. The neoplasm was interpreted to represent the malignant counterpart of benign angioma, of which the lip constitutes a common site of occurrence, in other words, *angioblastic sarcoma*. The structure in this case did not permit of differentiation between lymphatic and hemic vascular origin.

Histologically the degree of malignancy was relatively high, 3 to 4, if all sarcomata are classified into four groups for purposes of histologic prognosis, based chiefly on cellularity and anaplasia. According to criteria and terminology used by one of us[6] the malignancy index would be 4.

Course after Operation. The surgical wound healed and the patient was discharged from the hospital on June 26, 1935. The following October, he noticed a tumor of the left side of the neck. It was of subcutaneous location and increased in size gradually but rapidly. The patient was readmitted to the hospital on May 17, 1936 at which time a semisolid mass the size of a large grapefruit protruded from the left side of the neck, also overlying the mandible. Ulceration, sloughing and hemorrhage were noted on May 21, 1936. He was very weak and markedly emaciated. Death occurred on June 5, 1936.

During the period of rapid growth of this mass the patient had received Roentgen ray therapy without evident effect, a total of 1600 R. being distributed in divided doses from February 12 to March 23, 1936.

Autopsy. Extreme degrees of emaciation and dehydration were noted at autopsy. Most of the bones of the skeleton were clearly outlined.

A soft subcutaneous mass, 15 by 12 by 6 cm., extended from the left mastoid process to the midline of the neck anteriorly, and from the temple to a point 2 cm. above the clavicle. The lower portion, somewhat pendulous, overhung the clavicle. The convexity of the mass consisted of a nearly circular ulcerated crater, 11 cm. in diameter, which was filled with soft, spongy, gray and red tissue, in part semifluid, from which blood dropped freely on manipulation. The fingers could be introduced readily into crevices between the soft masses occupying the crater to a depth of 5 cm.

Dissection of the neck disclosed an invasive strand of neoplastic tissue, soft and grayish-white, resembling cerebral cortex, about 5 mm.

in diameter, extending from the mass along fascial planes downward, disappearing beneath the manubrium and clavicle. It apparently ended there as no intrathoracic extension could be traced. Tumor thrombi filled and distended the lumen of the external jugular vein to a diameter of 15 mm., but were not found in the subclavian and innominate veins or in the vena cava. The mandible was intact to palpation. No intraoral extension of the mass could be found nor any distortion or ulceration of the mucous membrane.

A wedge-shaped area of gray and hemorrhagic infarction, 2 cm. in diameter, was found at the base of the lower lobe of the right lung. A white tumor thrombus filled the axial blood vessel leading to the area. Except for bronchopneumonia of the lower lobe of the left lung, the autopsy revealed nothing further of note. No metastases other than that of the right lung could be found.

Microscopic studies revealed that the neoplastic tissue occupying the external jugular vein and the pulmonary arterial branch had a sarcomatous structure similar to that of the tumor removed from the lower lip in June, 1935.

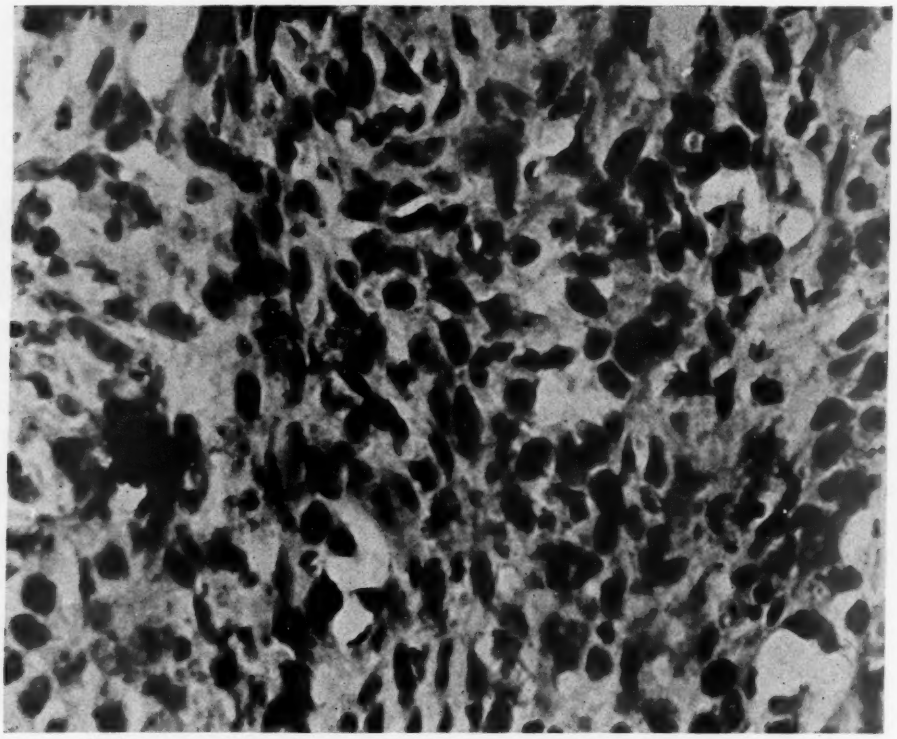

FIG. 3. High power photomicrograph of the tumor of the lip. Pleomorphism, mitosis, and the relationship of the cells to vascular channels are more clearly depicted.

COMMENT

The terminology employed in the classification of neoplasms varies considerably, rendering comparisons difficult. Sometimes bases of histogenesis and of morphology irrespective of histogenesis are used interchangeably in the same series, with the result that two or more instances of a given type of tumor are recorded under different designations, giving the impression that different types of neoplasms are referred to. For example, a "spindle-cell" sarcoma may well be a "neurogenic" sarcoma as well as one of fascial, vascular or osseous origin. One would believe angiosarcoma or angioblastic sarcoma to be a form rarely assumed by sarcoma of the lip, as judged by case reports. Yet Cases II and III of Cholnoky's paper, classified as spindle-cell sarcomas, and perhaps Case I, reported as round-cell sarcoma, have angioblastic features, as portrayed by the photomicrographs. Since angiomas scarcely are rare

among the benign lesions of the lip, one might expect that the malignant counterpart of these lesions would be encountered occasionally.

The resistance of this lesion to irradiation is manifest in that the tumor of the neck continued to grow rapidly during the course of radiotherapy and to the end of life, as if stimulated by it.

SUMMARY

A case of sarcoma of the lower lip is reported in a man 53 years old. The lesion was removed surgically two months after it was first noticed. Cervical metastasis appeared four months later, without recurrence at the operative site. In the course of the ensuing eight months this metastatic mass attained a diameter of 15 cm., and the overlying skin sloughed away, leaving a wide, deep, bleeding crater. Neoplastic thrombosis of the left external jugular vein and pulmonary metastases were found at autopsy. Death occurred fourteen months after the primary lesion was noticed and eight months after metastasis became manifest. Deep Roentgenray therapy was without apparent effect on the growth of the metastatic cervical mass.

REFERENCES

1. Cholnoky, Tibor de. *Am. J. Cancer*, 27: 548, 1934.
2. Simmons, C. C. *Am. J. Cancer*, 25: 621, 1935.
3. Geschickter, C. F., and Lewis, Dean. *Am. J. Cancer*, 25: 630, 1935.
4. Geschickter, C. F., and Keasbey, L. E. *Am. J. Cancer*, 23: 568, 1935.
5. Freilich, E. B., and Coe, G. C. *Am. J. Cancer*, 26: 269, 1936.
6. Pusch, Lewis C. *Radiology*, 16: 660, 1931.

BILIARY PERITONITIS WITHOUT PERFORATION AS A CLINICAL ENTITY

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A CASE of peritonitis of biliary origin without a perforation either of the gall-bladder or ducts seems worth recording, with a brief review of the literature.

CASE REPORT

The patient, a male of 38, reported on June 28, 1937, that for one month he had had irregular attacks of abdominal pain with no localization, nausea, vomiting or jaundice. His bowels were normal and the distress was not related to his meals.

On June 27, at 8 P.M. he had a sudden severe attack of excruciating pain in the mid-epigastrium. The severity of the pain, the shock, and the absence of other symptoms simulated coronary occlusion. One-half grain of morphine was administered, but gave no relief. After the hypodermic of morphine the patient vomited once. Eighteen hours after onset the pain radiated to the right lower quadrant and the patient was admitted to the hospital. He was then acutely ill, with severe epigastric distress and pain in the right lower quadrant. The abdomen was tender throughout with rebound tenderness most acute high in the right lower quadrant. There was no jaundice.

The temperature (rectal) was 101.4, the pulse 85, respirations 22, and blood pressure 140/90. The red blood cells numbered 5,050,000; hemoglobin was 100 per cent. The white blood cells were 12,700, with 78 per cent polys, 2 per cent eosinophiles, 11 per cent small lymphocytes and 9 per cent monocytes. Nuclear index was 3.2. Urine examination was negative; but the specimen was not examined for bile.

Acute appendicitis with peritonitis was diagnosed.

At operation 8½ ounces of dark brown fluid were found throughout the abdomen, but especially in the right lower quadrant, with greenish fibrin on the bowel. This suggested a ruptured ulcer, but palpation revealed an enlarged tense gall-bladder not surrounded by adhesions or omentum. The gall-bladder was dark, cylindrical and tense. There were no stones palpable nor was there any evidence of perforation. The cystic and common ducts were palpable, enlarged and indurated. The stomach and the duodenum were normal. The cystic duct was isolated and clamped near the common duct; it was found to be completely occluded with mushy material. Aspiration of the common duct produced dark bile; it was opened, irrigated with a catheter to the duodenum, and drained. The gall-bladder and appendix were removed.

Clinical and pathologic examination of gall-bladder and cystic duct revealed no perforation.

The gall-bladder measured 11 by 2.5 cm. and was covered with fibrinous purulent exudate. It was filled with a mushy, greenish, soft mass, with innumerable mulberry-like calculi. The mucous membrane was irregularly marked by areas of atrophic mucosa. The wall varied from 1 to 6 or 7 mm. and had the appearance of a chronic inflammatory process with acute inflammation. The appendix was grossly normal.

Microscopic examination of the gall-bladder showed a markedly thickened wall, with hyperemia and infiltration with polymorphonuclear leucocytes—a definite active infection probably grafted on an old process.

The patient made an uneventful recovery and was discharged on the eighteenth day after operation. Slight drainage persisted.

Clairmont and von Haberer,[4] Buchanan,[3] Tormey,[11] and Stenson[10] have reported similar cases and in each of these the clinician considered the fluid as bile. No perforation was demonstrated, although in no case has the fluid been examined chemically or bacteriologically and only in autopsied cases has it been possible to examine the entire biliary tract for perforation.

The appearance of the fluid is different from that of biliary ascitic fluid found in many other conditions. The absence of perforation seems substantiated in that most ruptured gall-bladders are localized and walled off. Wesson and Montgomery,[13] in sixteen cases of gall-bladder rupture, did not find generalized peritonitis in any at the time of operation.

Theories offered regarding the origin of the fluid include: filtration through the gall-bladder and bile ducts due to some undemonstrable pathologic change in their walls;[4] action of pancreatic juice making the wall permeable;[2] microscopic perforation with rupture of gall-bladder subsequently healed; rupture of intrahepatic bile canal; postperitoneal rupture of perforation of the common or hepatic duct.

Mentzer,[8] from a report of traumatic cases, concludes that sterile bile has not been proved to be a cause of death. He feels that death is due to pyogenic rather than chemical peritonitis when it occurs.

Harkins[6] produced bile peritonitis experimentally with ligation of the common duct and defundation of the gall-bladder. Two injected dogs died; three treated by aspiration also died. Harkins concludes that rapid death in bile peritonitis is due to shock, blood pressure fall and decrease in blood volume and increase in concentration.

Andrews and Henry[1] suggest that bacteria may play but a secondary rôle in disease of the gall-bladder and that other possible factors—mechanical, vascular, toxic and chemical—deserve more careful study.

Ravdin, Morrison and Smith[9] attempt to prove that bile ascitic fluid is relatively innocous because of low bile salt content. Whole bile injected into the dog produces death. They feel, therefore, that in bile peritonitis there must be an opening with direct communication with the biliary passages. Bacteria cannot be demonstrated as easily, even in pathologic gall-bladders, as they can be in normal intestine.[1,7,12]

CONCLUSIONS

1. Biliary peritonitis without perforation is believed to occur.

2. Experimental work does not substantiate it, however. The conditions in the human cannot be duplicated.

3. If fluid from the abdominal cavity and gall-bladder could be analyzed chemically and bacteriologically; and if these studies were correlated with pathologic examination of the gall-bladder, valuable data might be obtained. However, usually these patients are desperately ill and the efforts to save life overshadow the urge for experimental investigation.

4. If the patient's condition warrants it, the common duct should be drained.

REFERENCES

1. Andrews, E., and Henry, L. D. *Arch. Int. Med.*, 56: 1171, 1935.
2. Blad. Studien über Gallenperitonitis ohne Perforation der Gallenwege. *Arch. f. klin. Chir.*, 109: 101, 1916.
3. Buchanan, J. J., *Surg., Gynec. & Obst.*, vol. 26, March, 1918.
4. Clairmon, and von Haberer. Gallige Peritonitis ohne Perforation der Gallenwege. *Mitt. a.d. Grenzgeb d. Med. u. Chir.*, 22: 154, 1910.
5. Graham. Surgical Diagnosis. vol. 2, p. 619. Philadelphia, 1930. Saunders.
6. Harkins, H. N., Harmon, P. H., and Hudson, J. E. *Arch. Surg.*, 33: 576, 1936.
7. Lyon, B. B. V. *J. Lab. & Clin. Med.*, 17: 583, 1932.
8. Mentzer, S. H. *Arch. Surg.*, 29: 227, 1934.
9. Ravdin, I. S., Morrison, M. E., and Smyth, C. M., Jr. *Ann. Surg.*, 89: 867, 1929.
10. Stenson, W. *Am. J. Surg.*, 37: 334, 1937.
11. Tormey, A. R. *Wisconsin M. J.*, 33: 839, 1934.
12. Twiss, J. R., and Phillips, C. H. *Am. J. Digest. Dis. & Nutrition*, 2: 663, 1936.
13. Wesson, H. R., and Montgomery, T. R. *Proc. Staff Meet., Mayo Clin.*, 12: 500, 1937.

AVULSION FRACTURE OF THE LESSER FEMORAL TROCHANTER

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THE avulsion of the lesser femoral trochanter because of muscular violence is not a common fracture. According to McEachern and Jennings[1] (1931), there have been forty-eight reported cases in the literature. Since that time, seven additional cases have been recorded, bringing the total to fifty-seven, including the two cases reported below.

CASE I. J. S., a white boy, age 13, on November 24, 1937, complained of pain in the left hip and groin. He gave a history of sliding along on ice when suddenly he was seized with severe pain and fell down. He was carried home and subsequently sent to the hospital.

The pain in the left hip, the incapacity of the left leg and the *x*-ray film demonstrated an avulsion fracture of the lesser femoral trochanter.

FIG. 1. Roentgenogram showing avulsion fracture of the lesser femoral trochanter. (Case I.)

FIG. 2. Roentgenogram of the fracture three months after the injury. (Case I.)

Operation is not necessary in these cases. A plaster of Paris hip spica was applied with the knee flexed. The thigh was held in 90 degrees flexion, adducted and slightly internally rotated. After two weeks' hospitalization the patient was discharged with crutches. Six weeks after the accident the cast was removed. The result was excellent.

CASE II. A. R., colored, age 11, came under my observation September 25, 1938. There was a history of sudden severe pain in the right leg while running. This patient also was treated by means of a hip spica, leaving the hospital five days later, and making an uneventful recovery.

[1] MCEACHERN, J. S., and JENNINGS, H. N. Avulsion of the lesser trochanter of the femur. *Canad. M. A. J.*, 25: 449–450 (Oct.) 1931.

NEW INSTRUMENTS

A RUBBER-DAM COLOSTOMY MAT

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FOLLOWING the operative procedure of the formation of a colostomy, as part of a resection of the rectum, as a

FIG. 1. Colostomy mat before using.

permanent double-barrelled colostomy, or as a palliative Mikulicz operation, the clamp is usually removed in forty-eight hours postoperatively. At this time a psychic depression unfortunately occurs in most of the patients.

Any procedure which prevents the superimposed burden of a wound infection during postoperative convalescence is to be sought for. A simple device is presented to be used in the postoperative colostomy training until after the stitches have been removed and the wound has healed. The colostomy stump should purposely be left 1½ inches long; after the wound has healed and edema has resolved, it should then be cut down to ½ inch above the skin line. This usually can be done at the end of the second week.

A piece of measured tonsil snare wire is placed around the periphery of a circular sheet of rubber-dam, 10 inches in diameter. The edge of the rubber-dam is hemmed over the circular wire, giving us a rubber-dam mat.

All dressings are removed from the wound at the time of removal of the clamp. If agglutination of the stump to the skin is satisfactory, the wound and sutures are painted with tincture of iodine and a strip of iodoform gauze 2 inches wide is placed on the suture line and a sterile dressing is added. One or two strips of wide vaseline gauze covers the sterile dressing except for the opening through which the colostomy stump protrudes.

The sterilized mat (in alcohol) is then punctured in the center and the opening enlarged by traction, enough to enable the mat to be slipped over the colostomy stump and fit snugly. A 6 inch length of a half-inch strip of either iodoform gauze or vaseline gauze is wound around the base of the colostomy stump next to the rubber-dam mat. Additional layers of wide vaseline gauze are placed in coffer-dam arrangement to protect the colostomy. A drainage pad covers all the dressings.

We prefer to initiate colostomy training by enema the following day and we repeat

the enema daily at the same time. This soon trains the lower part of the colon to evacuate itself completely only at the time the enema is given. The disagreeable incontinence is avoided.

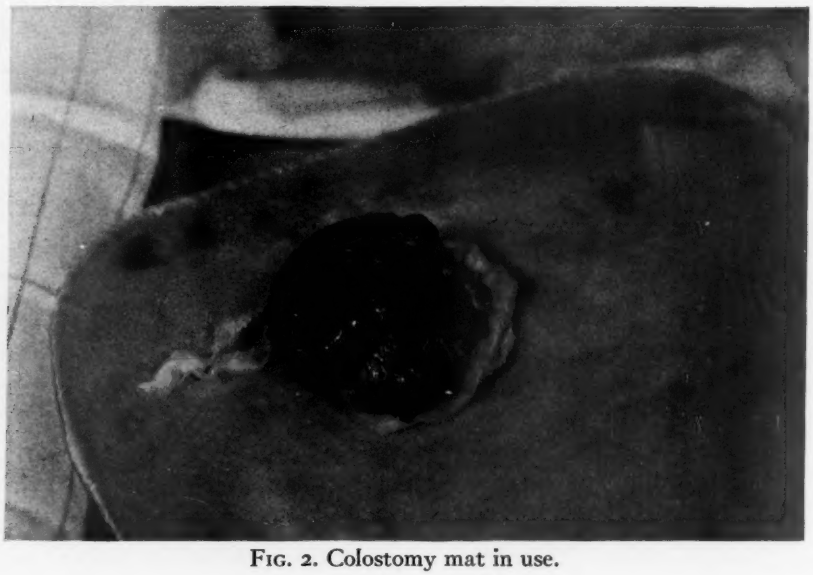

FIG. 2. Colostomy mat in use.

With each evacuation the rubber-dam mat with its fecal contents is lifted from the colostomy stump and after cleansing, the dressings and mat are replaced as described above. The abdomen is never soiled and the suture line is never contaminated.

With adherence to details on the part of the attendants, all wounds heal by primary union. Three or four rubber-dam mats should always be on hand and may be used repeatedly after cleansing and sterilizing, until the slit becomes too large to fit snugly, when they can be discarded.

CONCLUSION

1. A simple device, a rubber-dam mat, is presented to be used during postoperative training of the colostomy to insure primary healing of wound.
2. It avoids general soiling.
3. Confidence is established in the fact that there is no necessity for a colostomy bag.
4. It is a psychic aid in the management of the depression which follows the formation of an abdominal colostomy.

TRACTION ON THE ZYGOMATIC PROCESS FOR CERVICOVERTEBRAL INJURIES

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MINNEAPOLIS, MINNESOTA

IN the past, traction for cervical injuries by halters and various other methods by applying force under the chin or the occiput have proved to be very uncomfortable for the patient. Skeletal traction eliminates this disadvantage.

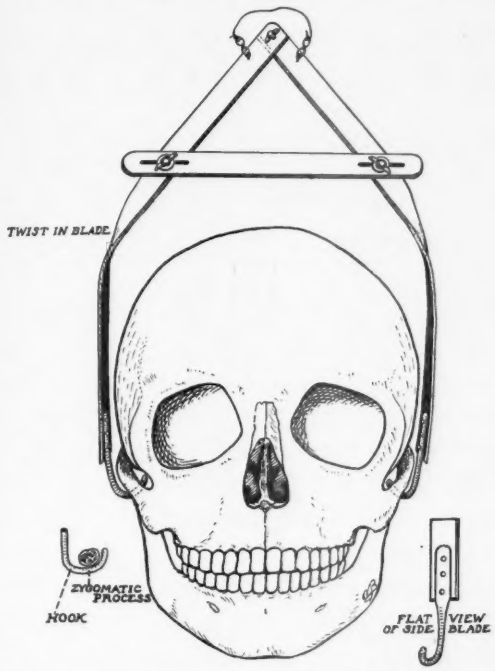

FIG. 1. Traction apparatus applied, showing relation of hook to zygomatic process.

The following apparatus may prove to be a better means of obtaining cervical traction, by making use of the zygomatic process on each side as an anchor post.

The apparatus represents the acme of mechanical simplicity. It consists of two separable side blades with a quarter twist just below the middle of the blade. These two blades are held together at the top by a removable lock bolt. Fastened to the bottom of each blade is a rustproof round hook with a square base wide enough so the outer prong does not compress the skin and soft tissue over the zygomatic process. Where it is feared this will not prove effective, a flat hook $\frac{3}{16}$ to $\frac{1}{4}$ inch wide can be attached. The point of the inside prong or bar is made round and smooth, that no soft tissue injury can be produced. The removable cross bar, which prevents the blades from spreading or coming together, is held in place by two removable bolts. All bolts have winged nuts to facilitate tightening.

The method of inserting the hooks beneath the zygomatic process is simple. With the usual aseptic procedures, 2 per cent procaine is injected under the zygomatic process at its midportion, just in front of the tubercle. The needle is then tilted up as much as possible and procaine injected along the inner surface of the process. A very small nick is made in the skin, enough to allow the hook to pass through. A button hooklike instrument with a round, smooth, dull point is then inserted and directed under and upward around the zygomatic process, always hugging the bone. This grooves a pathway for the hooks which are to follow. The same is done on the other side. The sterilized tractor hooks are then carefully inserted. The top bolt is then put on, followed by the cross bar which is adjusted so the inner post of the hooks are in immediate contact with the inner surface of the zygomatic process. The instrument is now set for traction.

As no pin is drilled or set into the cortex of bone, there is less danger of osteomyelitis, and because the traction hooks or bars are pulling on hard, undamaged bone there is less chance of bone erosion, loosening or cutting through as in the intra-osseous pin or screw.

[From Fernelius' Universa Medicina; Geneva, 1679.]

BOOKSHELF BROWSING

SMOLLETT, LIND AND ANSON IN 1739

THEIR COMMON CONTRIBUTION TO THE CONTROL OF SCURVY

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IN 1739, just 200 years ago, England entered upon a period of warfare with Spain and France. Seventy-six years of intermittent conflict followed. How the ebbing power of Spain and the flood tide of French imperialism were stemmed, and how these empires surrendered vast tracts of their domains as the price of peace—these are the facts of history. England's mastery at sea and economic strength at home won victories not to be bought by Spanish gold, victories of more enduring character than Napoleon's brilliant battles. The year 1739 was momentous in the history of the British Empire, for it was the year in which the birth pains of that empire set in. The political happenings culminated in the establishment of British rule over one-fourth of mankind.

1739 was also a significant year in the history of medicine. Events in the lives of three men in that year led to man's effectual control over the most dreaded of all old-time sea diseases, the scurvy. In 1739, three men, unknown to each other, made important changes in their lives, important for posterity. One went to London. One went to sea. One returned from the West Indies to take command of an expedition fitting out for the Far East. Their names were Tobias Smollett, James Lind and George Anson. Their achievements in the fields of English literature, medical science and navigation all contributed to the immense improvement in the health of seamen which followed mastery of scurvy in the British Royal Navy, a major triumph of eighteenth century medicine.

England formally declared war against Spain October 23, 1739, but informal war had been in progress many years. Two hundred years ago, just as today, international "incidents" had political value out of proportion to their probable authenticity. Whether or not that charming fellow, Juan de Leon Fandino, captain of His Catholic Majesty's guardacostas in April, 1731, halted and boarded the *Rebecca*, brig, of Glasgow, bound from Jamaica to London, and whether or not Captain Fandino, in protest against an inhospitable reception, clipped an ear from Captain Jenkins's head and made remarks derogatory to the British throne—even today no one can be sure. The fully established, more important fact is that in 1738 Captain Jenkins appeared before Parliament, told his story, offered his ear in evidence, and so excited public indigna-

tion that Walpole was reluctantly compelled to sanction measures of reprisal. In 1739 these measures led to open war,

A

TREATISE

OF THE

SCURVY.

IN THREE PARTS.

CONTAINING

An inquiry into the Nature, Cauſes, and Cure, of that Diſeaſe.

Together with

A Critical and Chronological View of what has been publiſhed on the ſubject.

By *JAMES LIND*, M. D.

Fellow of the Royal College of Phyſicians in *Edinburgh*.

EDINBURGH:

Printed by SANDS, MURRAY, and COCHRAN.

For A. MILLAR, in the Strand, *London*.

MDCCLIII.

FIG. 1. Title Page of Lind's Treatise of the Scurvy, 1753. The date is often misquoted. (Courtesy of New York Academy of Medicine.)

the War of Jenkins's Ear, as it was lightheartedly named. No substantial treaty of peace was to be written until 1815. Events of 1739 were a prelude to suffering and death in the steaming jungles of India, on the wooded slopes of America, on the sands of Egypt and on the heights of Quebec. Events of 1739 bred those of 1776, with Bunker Hill, Princeton, Saratoga and Valley Forge. But it was not on land but on sea that the fate of empires was determined. The Board of Admiralty, in 1739, had already drawn its plans.

The scheme was olympian in scope. In the Caribbean, in the Pacific and in the South China Sea, attack was to be made on Spanish colonies whose remoteness hitherto had been their chief protection. Two of these three expeditions eventually set sail, the one to the Caribbean under Vice-Admiral Edward Vernon departing August 3, 1739. Admiral Vernon was on no mission of appeasement. His orders, dated July 19, 1739, three months before formal declaration of war, instructed him "to destroy the Spanish settlements in the West Indies and to distress their shipping by every method whatever." That was undeclared war with grim vengeance! Destined to serve under Admiral Vernon in the humble capacity of surgeon's mate, was England's greatest physician-novelist, Tobias Smollett, but in 1739 he was unaware of that destiny.

Tobias Smollett was born in the valley of Leven in Scotland in 1721. In 1736 he was apprenticed to Dr. John Gordon of Glasgow, where he was a fellow student of William Hunter. For three years he pursued his studies. Then, in 1739, came a momentous decision: he would go up to London, mecca of young Scotsmen on the make. Perhaps Smollett's motives are revealed in the persuasive arguments addressed to Roderick Random by that worthy and vigorous surgeon, Mr. Launcelot Crab: "I am surprised that a young fellow like you discovers no inclination to push his fortune in the world. Before I was of your age, I was broiling on the coast of Guinea. Damme! what's to hinder you from profiting by the war, which will certainly be declared in a short time against Spain? You may easily get on board of a king's ship in quality of surgeon's mate, where you will certainly see a good deal of practice, and stand a good chance of getting prize money."

It was the first day of November, 1739, when Roderick Random took his leave of Mr. Crab and set out with carriers bound

for Newcastle. The year is known to be that of Smollett's London journey; no doubt the day and month are those given, and no doubt the inventory of Rory Random's kit is the tally of Smollett's worldly wealth at this important juncture in his career: "One suit of clothes, half a dozen of ruffled shirts, as many plain; two pairs of worsted, and the like number of thread stockings; a case of pocket instruments, a small edition of Horace, Wiseman's *Surgery*, and ten guineas in cash; for which Crab took my bond, bearing 5 per cent interest." One item known to be carried by Smollett is missing from the list: the manuscript of his first magnum opus, a tragedy, hopelessly bad, which he had written when he might better have been studying his Wiseman.

It was in 1739 that Smollett made this important journey, and when, in October, 1740, Rear Admiral Sir Chaloner Ogle sailed for Jamaica with men and supplies for Admiral Vernon, one of his ships carried as surgeon's second mate the raw young Scot whose immortal record of his experiences at sea took shape in 1748 as the first great English maritime novel, *The Adventures of Roderick Random*.

Roderick Random was more than a narrative. In describing the cockpit, the sickbay, the rationing, the treatment of the sick and the squalor and horror below decks, Smollett revealed the appalling indifference of the Admiralty to the welfare of the men and the urgent need for hygienic reform. Sent out between the covers of a best seller, Smollett's revelations had wider circulation than if they had been issued as a tract on naval hygiene, and they were more effective, because they were read. By exposing the crudities of naval hygiene in the blaze of his satire, Smollett made an important contribution to its ultimate amelioration. Good general hygiene is a most potent force in the control of scurvy.

Smollett's contribution to the control of scurvy by propaganda for the general improvement of naval hygiene was incidental to his main purpose: to sell a novel. Another Scotsman was to have as his first aim, not the writing of a novel, but the composition of a volume on scurvy that would serve as a record of his observations and the observations of all earlier writers on the subject. That purpose had not been conceived in 1739, but in that year this other Scotsman, James Lind, went to sea, and out of his experiences there that purpose and its fruition came.

James Lind was born in Edinburgh October 4, 1716. At the age of 15, he, like Smollett, was booked apprentice. Smollett worked three years for Dr. Gordon; Lind served Mr. George Langlands, surgeon, of Edinburgh, the full seven-year term. Then, in 1739, with little prospect of advancement in Edinburgh, for reasons perhaps resembling those which influenced Roderick Random, Lind went up for a surgeon's mate's warrant, received it, and embarked in one of His Majesty's ships.

Lind remained in the Royal Navy for nine years, whereas Smollett spent only two. By 1748, the year of Lind's retirement, Smollett had been ashore six years; and had finished *Roderick Random*. Lind, too, had experiences to place on record, but not in the form of fiction. In 1753 appeared *A Treatise of the Scurvy*, a work that remains today an enduring monument to Lind's fame, as important to science as *Roderick Random* is to literature. Lind sifted a kitchen midden of sea lore, medical dogma, false deduction and half expressed truth. Guided by his own observations and by his practical knowledge of the disease, he laid the sound foundations of modern diagnosis and treatment. He was not the first to propose the use of citrus fruit juices in treatment of scurvy (he stated himself that "their experienced virtues have stood the test of near 200 years") but he was the first to review as completely as he could all previous work on the subject and compare other authors' conclusions with his own by the test of experimentation. After Lind, no further demonstration was needed

that orange and lemon juice were superior to all other remedies in the treatment and prevention of scurvy.

"On the 20th of May 1747, I took twelve patients in the scurvy, on board the Salisbury at sea. Their cases were as similar as I could have them. . . . They lay together in one place, . . . and had one diet common to all . . .

"Two of these were ordered each a quart of cyder a-day. Two others took twenty-five gutts of *elixir vitriol* three times a-day . . . Two others took two spoonfuls of vinegar three times a-day . . . Two of the worst patients . . . were put under a course of sea water. Two others had each two oranges and one lemon given them every day. The two remaining patients, took the bigness of a nutmeg three times a-day, of an electuary . . . made of garlic, mustard-seed, *rad. raphan*, balsam of *Peru*, and gum myrrh.

"The consequence was, that the most sudden and visible good effects were perceived from the use of the oranges and lemons; one of those who had taken them, being at the end of six days fit for duty . . . The other was the best recovered of any in his condition; and being now deemed pretty well, was appointed nurse to the rest of the sick."

In medical literature there are few experiments more convincing than that one of Lind's. Many years were to pass, and Lind himself was to be in his grave before the simple measures he proposed were to find favor with the august Lords of the Admiralty through the influence of Sir Gilbert Blane. When, in 1795, Lind's observations were applied, and an order was given that lemon juice was to be issued daily on all ships after two weeks on salt meat, scurvy was banished from the British navy.

The first edition of *A Treatise of the Scurvy* is dedicated to the Right Honorable George, Lord Anson "who, as a just reward for the great and signal services done to the British nation, does now preside over her naval affairs." In the introduction Lind acknowledges that it was publication of the account of Anson's circumnavigation of the globe which first led him to think of writing the book which made him immortal.

George Anson was born in Staffordshire in 1697. At the age of fifteen he entered the navy and ten years later, in 1722, was given his first independent command. Seventeen years of active service followed. In 1739 his great opportunity came. By the Admiralty plan, a man was needed to command one of the squadrons which was to be sent out to fight the Spaniard on the other side of the world. Anson was chosen and recalled from his station in America to play a rôle in which he was to capture and forever hold the imagination of a maritime race. His mission, as finally conceived, was a dangerous one. He was to round the Horn, and, while Vernon operated in the Caribbean, he was to "distress and annoy the King of Spain and his subjects" along the west coast of South and Central America. The eight vessels of Anson's squadron put to sea September 18, 1740. Anson was a brilliant leader. English seamen were superior to those of any other nation. Three ships survived the Horn, but after rounding it, they were no longer effective vessels of war. No extensive campaign could be undertaken, and all thought of the substitution of English for Spanish rule in Central and South America was at an end. Scurvy had taken its toll. Let Chaplain Richard Walter describe conditions aboard Anson's flagship:

"Soon after our passing Straits Le Maire, the scurvy began to make its appearance amongst us; and our long continuance at sea, the fatigue we underwent, and the various disappointments we met with, had occasioned its spreading to such a degree, that at the latter end of April there were but few on board, who were not in some degree afflicted with it, and in that month no less than forty-three died of it on board the *Centurion* . . . in the month of May we lost near double that number: and as we did not get to land till the middle of June, the mortality went on increasing, and the disease extended itself so prodigiously, that after the loss of above two hundred men, we

could not at last muster more than six foremast men in a watch capable of duty; . . . to so wretched a condition was a sixty-gun ship reduced, which had passed Straits Le Maire but three months before, with between four and five hundred men, almost all of them in health and vigour . . . "

Two hours to trim the sails; four hours to heave the anchor cable up and down, and then unable to start it because the capstan was so weakly manned! A long period of rest and recuperation at Juan Fernandez restored many to health, but those who had died could not be replaced, and of 961 men comprising the original crews of the three vessels which reached Juan Fernandez, only 335 were living in September, 1741, most of the 626 dead having succumbed to scurvy.

Anson returned, laden with booty, in 1744 and Chaplain Walter, in 1748, the year of *Roderick Random* and the year of Lind's return to Edinburgh, published *A Voyage Round the World in the Years 1740–1–2–3–4 by George Anson, Esq., now Lord Anson*, which ran to four editions in the first year. The ravages of scurvy were described with overwhelming force, while the dramatic events of Anson's voyage aroused intense popular interest. Lind was only one of many who must have been stirred by the pitiful account of brave men crippled and destroyed by scurvy. With them died English dreams of a South American empire, but their deaths and Chaplain Walter's telling made men aware of the cruel extent of scurvy's ravages at sea and directly led to Lind's great treatise.

Anson returned to occupy the highest posts in the Admiralty and became Admiral of the Fleet before his death in 1762. He was First Lord when Lind dedicated his *Treatise* to him; as First Lord in 1758 he appointed Lind physician to the new Naval Hospital at Haslar, a post Lind occupied for many years. Lind died in 1794, at the age of 78. Smollett pursued that fickle hussy, literary fame, with variable success. Never was he further from her favor than when, in 1769, he produced *The History and Adventures of an Atom*, which is mentioned here only because of the bitter lampoon of the dead Admiral Anson under the soubriquet of Nin-kom-poo-po. Smollett was, however, an ailing man and he survived only long enough to finish *The Expedition of Humphrey Clinker* which appeared in 1771.

Smollett, Lind and Anson live as long as books are treasured and read. Two hundred years ago their destinies took definitive shape. Their single important common experience, naval service, gave us a novel, prototype of naval fiction; a treatise, greatest of its kind; and a travel narrative, yet unsurpassed, all three of which contributed to one of man's grandest medical victories, control of scurvy.

REFERENCES

Smollett

Drinker, C. K. Doctor Smollett. *Ann. M. Hist.*, 7: 31, 1925.

Jones, C. E. An essay on the external use of water by Tobias Smollett, *Bull. Inst. Hist. Med.*, 3: 31, 1935.

Lind

Rolleston, H. D. James Lind, pioneer of naval hygiene. *J. Roy. Nav. Med. Serv.*, 1: 181, 1915.

Stockman, Ralph. James Lind and scurvy. *Edinburgh M. J.*, 33: 329, 1926.

Anson

Anson, Walter Vernon. The Life of Admiral Lord Anson, the Father of the British Navy, 1697–1762. London, 1912. John Murray.

Walter, Rev. Richard. A Voyage Round the World in the Years 1740–1–2–3–4 by George Anson, Esq., now Lord Anson. London, 1748.

General Sources

Clowes, W. L. The Royal Navy, vol. 3. London, 1898. Sampson Low, Marston.

Dictionary of National Biography.

Vogel, Karl. Scurvy, the plague of the sea and the spoyle of mariners. *Bull. New York Acad. Med.*, 9: 459, 1933.